Trends and social inequalities in cardiovascular risk factors

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Declaration

Except where it is indicated otherwise, the work in this thesis is my own, and is based on original research conducted at the National Centre for Epidemiology and Population Health, The Australian National University, Canberra.

The material in chapter 4 'Trends in cardiovascular risk factors in Australia' formed the basis for an article published in the *Medical Journal of Australia* with Dr Paul Magnus, Medical Director of the National Heart Foundation of Australia. My involvement included intellectual input, all aspects of the statistical analysis and the initial draft of all sections of the paper.

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This revised version of the thesis incorporates minor amendments suggested by the examiners, and corrects typographical, grammatical and similar errors.

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I am grateful to Dr Ralph Reader who had the vision to initiate the Risk Factor Prevalence Study, and to Dr Bob Hodge who carried it to fruition. I am grateful also to the study's Management Committee for their permission to analyse the data. I am especially indebted to the 20,000 respondents who gave of their time to attend the study's survey centres for cardiovascular risk factor assessment, and to the staff in the clinics who collected the data.

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Abstract

Cardiovascular disease places a heavy burden on Australians in terms of illness, disability and economic cost, primarily from coronary heart disease and stroke. The disease is highly preventable. Lifestyle patterns and biomedical risk factors are known to make significant contributions to cardiovascular mortality and morbidity, and evidence suggests that social and economic factors have effects which are not entirely explained by differentials in lifestyle behaviour and risk factor levels. Australia's capacity to develop cost-effective and equitable strategies for community prevention and treatment is limited by a lack of information about the distribution of risk factors, particularly among lower socioeconomic groups. The study of trends and socioeconomic inequalities in risk factors for cardiovascular disease has the potential to yield considerable benefits in the national endeavour for social justice in cardiovascular health.

The thesis presented here is that the cardiovascular risk factor profile of Australians improved during the 1980s, consistent with falling death rates, but important social inequalities remain. Trends and inequalities in cardiovascular mortality and risk factors are examined using death registration data and data from a series of risk factor prevalence surveys conducted in Australia during the 1980s. Using a common protocol, this series of surveys collected data on major biomedical risk factors and associated behaviours for over 22,000 respondents. Few countries have a similar national data resource.

First, an analysis of recent changes in the cardiovascular risk factor profile of Australian adults shows that reductions in cigarette smoking and blood pressure are likely to have contributed to the falls in cardiovascular mortality. Little change has occurred in blood lipids or in patterns of exercise during leisure time. Changes in self-reported dietary behaviour have been consistent with health education messages. A significant trend towards greater body fatness is identified which requires greater attention.

Next, variation in blood pressure measurement technique between survey centres is examined in order to verify the falls in blood pressure observed in the trend analysis. The Australian experience is used to demonstrate that certain measurement characteristics may inflate estimates of the prevalence of high blood pressure, and could contribute 0.85 mmHg to time trends or geographic differences in mean systolic blood pressure. The effect on the trend

analysis is estimated at 0.5 mm Hg, or 12% of the estimated fall in systolic blood pressure, and it is concluded that the observed falls represent real decreases in the average level of blood pressure among Australian adults.

Risk factor levels among Australia's immigrants are then examined to see whether beneficial profiles are a potential explanation of their significantly lower cardiovascular mortality compared with the Australian-born. Although significant differences are identified between immigrant groups and the Australian-born, overall results suggest that profiles of risk factors commonly accepted as determinants of cardiovascular disease are an insufficient explanation of immigrants' lower cardiovascular mortality. It is also demonstrated that the acculturation process affects the risk factor profile of immigrant groups differently.

Trends and socioeconomic inequalities in cardiovascular mortality are then compared with movements in major risk factors. Using occupation as the indicator of socioeconomic status, it is shown that men in manual occupations are 35% more likely to die from coronary heart disease than men in professional occupations, and 60% more likely to die from stroke. Their 5-year risk of a coronary event is 30% higher. It is demonstrated that the mortality inequalities of the 1970s persisted into the nineties and that the distribution of blood pressure and cigarette smoking in society contributed most to socioeconomic differentials and to declines in coronary risk.

By using educational attainment to indicate socioeconomic status, trends in socioeconomic inequalities in risk factors are examined for men and women. The lower socioeconomic group has improved its risk factor profile, but its relative disadvantage compared with the higher socioeconomic group persists. These results are generally consistent with parallel declines in mortality. It is demonstrated that significant behaviour change can occur simultaneously across the socioeconomic spectrum, not just in higher socioeconomic groups.

The summary chapter presents a framework to illustrate the relationships between cardiovascular health and circumstances and discusses reasons for the major trends and inequalities. It is concluded that health promotion activities in Australia have been effective in reaching the lower socioeconomic groups but the challenge to reduce inequalities remains. Implications for future health promotion are discussed and recommendations are made for future monitoring.

List of Publications

Bennett SA. Socioeconomic trends in death from coronary heart disease and stroke among Australian men. *Int J Epidemiol* (in press).

Bennett SA (1995) Australian trends in socioeconomic inequalities for cardiovascular risk factors. *J Epidemiol Community Health* 49:294-313.

Bennett SA and Magnus P (1994) Trends in cardiovascular risk factors in Australia. Results from the National Heart Foundation's Risk Factor Prevalence Study. *Med J Aust* 161:519-527.

Bennett SA (1994) Blood pressure measurement error: its effect on cross-sectional and trend analyses. *J Clin Epidemiol* 47:293-301.

Bennett SA (1993) Inequalities in risk factors and cardiovascular mortality among Australia's immigrants. *Aust J Public Health* 17:251-261.

Contents

Chapter One

Introduction

1.1 A brief history

During the 1950s and early 1960s in Australia, death rates for coronary heart disease almost doubled, increasing by 93% in men and by 101% in women (d'Espaignet 1994). This upward trend was reversed during the late 1960s and coronary mortality has continued to fall dramatically with the present levels being comparable with those of the fifties. The National Heart Foundation of Australia was quick to recognise the emergence of the downward trend (Reader 1972), the importance of reducing risk factors (The Committee on Diet and Heart Disease 1974), and the potential role of improved emergency care, advances in cardiac surgery, new and improved drugs for hypertension, and changes to rehabilitation (Reader 1979). To what extent the decline in mortality in Australia was due to better survival after a heart attack or to a fall in disease incidence due to improvements in the risk factor profile was not known. To help answer this question, heart attack registers in Perth (Martin et al. 1989) and Newcastle (Dobson et al. 1988) were used to monitor the incidence of the disease and the prevalence of cardiovascular risk factors was monitored by a series of cross-sectional surveys in Australia's capital cities, commencing in 1980 (Hodge 1984).

It is recognised that lifestyle patterns and biomedical risk factors significantly influence cardiovascular mortality and morbidity (Mitchell et al. 1980; Beaglehole et al. 1989; Hill et al. 1993; Martin et al. 1989; Beaglehole 1990; Goldman and Cook 1984). There is also evidence that social factors have an effect which is not entirely explained by differentials in lifestyle behaviour and risk factor levels (Marmot et al. 1984). Increasing recognition has been given to the role played by structural social factors such as the level of economic resources available to individuals, education, living conditions, working conditions and social support (National Health Strategy 1992a). Cultural influences may also be important, for example, in contributing to lower coronary mortality among many of Australia's immigrant groups. The Australian Government has acknowledged that there is too little information about the pattern of risk factor levels in high-risk groups, such as low socioeconomic groups, and this limits Australia's capacity to develop cost-effective and equitable strategies for community prevention and treatment

(Commonwealth Department of Human Services and Health 1994). It follows that the study of trends and socioeconomic inequalities in risk factors for cardiovascular disease, the subject of this thesis, is important for developing, monitoring and evaluating public health interventions.

Much of the raw material for the analyses contained in this thesis was provided by the series of surveys conducted by the National Heart Foundation during the 1980s which comprise the Risk Factor Prevalence Study. All surveys were undertaken in conjunction with the (then) Commonwealth Department of Health. The Australian Institute of Health and Welfare contributed significantly to the conduct of the 1989 survey. Each survey included a physical examination component which collected anthropometric measurements, blood pressure and blood analysis data. No other Australia-wide probability surveys have collected information on such a broad range of biomedical and behavioural risk factors and few countries have a comparable national resource.

My involvement with the Study began in 1979 at the first meeting between the (then) Director of the National Heart Foundation, Dr Ralph Reader, and officers of the (then) Commonwealth Department of Health to discuss support by the Department for the initiative. My official role was Study statistician, but I soon became involved in all aspects of the study with the National Study Directors, Dr Victor Pippett and Dr Tom Sheehan (1980 survey), and Dr Paul Magnus (1983 and 1989 surveys). This included input into all areas of survey development and operations, including formulation of study objectives, survey design and sampling methods, questionnaire and form design, pilot testing, survey operations and staff training, drafting survey protocols and coding manuals, design of data verification and editing procedures, data analysis and report writing. I applied for and was granted approval to access the datasets for the purposes of this thesis in 1990.

1.2 Structure of the thesis

This study develops the proposition that the cardiovascular risk factor profile of Australians improved during the 1980s, consistent with falling death rates, but that this improvement differed among social groups and important social inequalities remain.

Chapter 2 is a literature review which provides a context for the development of the thesis. The review is necessarily selective because of the wealth of material that has been published.

An overview of methods used in the Risk Factor Prevalence Study is given in chapter 3. It includes response rates and the sample distribution for each survey, details of the measurement procedures and definitions of risk factors, and discusses issues related to data analysis.

Chapter 4 examines the hypothesis that the risk factor profile of Australians improved during the 1980s in line with the decreasing mortality rates for cardiovascular diseases. Health education messages of the 1980s advocated the importance of a healthy lifestyle, stopping smoking, taking regular exercise, eating a `healthy' diet, drinking alcohol in moderation and watching one's weight—all of which can affect cardiovascular risk. The analysis examines the (presumed) effect that public education messages such as these, regulatory measures, and changes in medical practice, had on the risk factor profile of Australians. The chapter also identifies changes in levels of risk factors which might be associated with the continuing fall in cardiovascular mortality rates.

Chapter 5 examines whether the trends in blood pressure identified in chapter 4 were influenced by variation in blood pressure measurement technique between surveys. Normal mercury sphygmomanometers were used to measure blood pressure in each of the three surveys, the final reading being the average of two consecutive readings taken five minutes apart. Although staff had been given training in the taking of blood pressure according to a standard protocol, there appeared to have been considerable variation in measurement technique between local centres and between surveys. Basic monitoring procedures had detected variation in the proportion of readings with zero as the final digit and variation in the mean difference between the two consecutive readings. It was important to assess the effect of this variation, especially since changes in mean blood pressure and in the prevalence of high blood pressure over time tend to be relatively small.

Chapter 6 assesses whether a beneficial risk factor profile might explain the lower mortality rate from cardiovascular disease which is experienced by many immigrant groups in Australia. The analysis is the first to compare systematically the levels of a wide range of biomedical and behavioural risk factors among a relatively detailed classification of countries of birth. The effect of length of stay in Australia on risk factor levels is also examined.

Using occupation as the indicator of socioeconomic status, chapter 7 examines several hypotheses including: that the socioeconomic inequalities of the 1970s have continued into the 1990s; that inequalities in mortality are consistent with those in risk factors; and that socioeconomic groups which improved their risk factor profiles the most also experienced greater improvements in mortality. The analysis is for men only. The lack of an occupation code on the death

certificate for much of the time series, and poor quality data thereafter, precludes analysis for women. Chapter 8 documents trends in socioeconomic inequalities in cardiovascular risk factors for men and women by using educational attainment as the indicator of socioeconomic status. It examines the hypothesis that socioeconomic groups responded differently to the health promotion activities of the 1980s and experienced different trends in biomedical risk factors.

The stimulus for chapters 7 and 8 was provided by the Black report (Black 1980) and subsequent analyses of British mortality data (Whitehead 1987), which showed that the gaps between the social classes in Britain were widening for most diseases, including coronary heart disease and stroke. Analysis of Australian mortality data of the 1970s also suggested widening inequalities (Dobson et al. 1985) which raised the question of whether a similar phenomenon might be found for risk factors. Very little has been published on trends in risk factor inequalities. The Risk Factor Prevalence Study is the only data source in Australia, and one of few internationally, which can be used to explore such issues.

The final chapter discusses the major findings of the thesis and makes recommendations for future monitoring activity in Australia. Chapters 4, 5, 6, and 8 have formed the basis for four published journal articles.[1] A paper based on chapter 7 has been accepted for publication.[2]

1.3 Relevance of the thesis

In recognition of the burden of cardiovascular disease on Australian society and the potential of prevention to effect a positive influence, the Federal government has chosen cardiovascular disease as one of four focus areas for the development of national goals, targets and strategies. The principle of social justice is reflected in its intention to reduce the level of health inequalities between population groups. The report notes that there is little risk factor prevalence information about high-risk groups and acknowledges that this

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Bennett SA (1993) Inequalities in risk factors and cardiovascular mortality among Australia's immigrants. *Aust J Public Health* 17: 251-261.

[2] Bennett SA. Socioeconomic trends in death from coronary heart disease and stroke among Australian men. *Int J Epidemiol* (in press).

deficiency limits 'Australia's capacity to make national decisions for the cost-effective allocation of health funds, planning for preventive and treatment services and targetting of priority groups' (Commonwealth Department of Human Services and Health 1994, p94). The report identifies non-English speaking immigrants and socioeconomically disadvantaged Australians as priority groups[1]. The analysis of risk factor levels among these two population groups form an integral part of the development of the thesis.

The reduction of risk factors and their inequalities is clearly an important element in the Government's strategy to improve the health of Australians. Results from the thesis provide a stronger reference base from which to develop and implement programs to improve the cardiovascular health of the Australian population and to reduce health inequalities in these priority groups. The findings for Australia are both timely and practical.

Internationally, the thesis results contribute to repository of data on recent trends in risk factors. In reviewing the status of the three most current US cardiovascular disease risk reduction studies,[2] a recent editorial for the American Journal of Public Health recognised that a:

> '... major contribution of these studies is the documentation of secular declines in cardiovascular risk factors among population-based samples of women and men during the 1980s' (Winkleby 1994).

This thesis provides such data for Australian women and men, by age group and for socioeconomic strata. The reasons behind the trends and social inequalities, and their implications for health promotion, are also discussed, and recommendations are made for future monitoring.

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[1] The report also identifies Aboriginal and Torres Strait Islander peoples as a priority group, however, this population group was not separately identified in the Risk Factor Prevalence Study and therefore could not be considered in this thesis.

[2] The Standford Five-City Project, the Minnesota Heart Health Program, and the Pawtucket Heart Health Program.

Chapter Two

Background

2.1 Cardiovascular disease in Australia

The disease

Cardiovascular disease includes all diseases of the heart and the circulatory system. The disease mostly results from diminished or impeded blood supply to the heart, brain or leg muscles and has been traditionally regarded as primarily a degenerative disease, that is, an outcome of the ageing process. Evidence now shows that it is closely associated with lifestyle and environmental factors, which are linked to social and economic conditions (National Health Strategy 1992a). This is significant because it means that early death and disease are largely preventable.

The process of atherosclerosis, which damages and can ultimately block the blood supply, is a progressive condition which often begins in childhood or youth. Atherosclerosis is most common in countries like Australia where a high fat national diet is associated with relatively high average levels of blood cholesterol. Cigarette smoking and high blood pressure accelerate the rate at which atherosclerosis develops. A high fat diet and smoking increase the tendency for a clot to form, and smoking may increase the chances of the heart developing a disturbed rhythm. In addition, both smoking (through the effects of nicotine and carbon monoxide) and long-term high blood pressure increase the heart's demand for oxygen from the blood while reducing the supply (Taskforce on Cardiovascular Disease 1986).

Mortality

Cardiovascular disease accounted for 44% of all deaths in 1993, having peaked at 58% in 1968. Its most common manifestations are coronary heart disease and stroke which accounted for 56% and 23% respectively of all deaths from cardiovascular disease. Coronary heart disease remains the greatest single cause of death in Australia, causing approximately as many deaths each year as occur from all forms of cancer together.

Death from coronary heart disease occurs more frequently in men than women of the same age. In 1992, the ratio of male to female age standardised death rates was 1.80. For stroke the ratio was 1.12 (Bennett et al. 1994). Although the

death rate from coronary heart disease increases greatly with age, it is not simply a disease of old age. In 1992, over 4,700 such deaths occurred in people aged less than 65, which is, for example, considerably more than the 1,730 deaths from motor vehicle accidents in the same age group (Bennett et al. 1994).

Death rates from coronary heart disease peaked in the late 1960s and have fallen dramatically since (d'Espaignet 1994). The decrease in the death rate from stroke has occurred steadily since the mid-1970s. Cardiovascular death rates have fallen at a greater rate than 'all cause' mortality (Table 2.1). Falls have occurred across all adult age groups; however the falls among older age groups, while still substantial, have been less than among younger age groups (Bennett et al. 1994).

TABLE 2.1 ***Annual rates of change in cardiovascular disease mortality rates for the period 1981-1992, Australia***

Cause of death	Males	Females
	%	%
Coronary heart disease	-3.6	-2.6
Cerebrovascular disease	-4.0	-4.7
All cardiovascular disease	-3.8	-3.3
All causes	-2.1	-1.6

Of 23 selected countries for which the World Health Organization provides standardised death rates for the age group 25-74, fifteen countries reported lower death rates from coronary heart disease than Australia (ie. Australia ranked 16th lowest), reinforcing the need to reduce mortality from coronary heart disease even further. Australia's death rates for stroke were ranked 6th lowest (Australian Institute of Health and Welfare 1994, p90). The fall in coronary heart disease death rates observed in Australia since the 1960s has been closely paralleled in the United States, Finland, New Zealand, Israel, Canada and Japan (albeit from a lower level in Japan). The decline began more recently in the United Kingdom and other European countries (Thom 1989; Beaglehole 1990).

Morbidity

It is estimated that there were 22,000 heart attacks among Australians aged 20 to 69 in 1992 of which 16,700 were first attacks (National Heart Foundation of Australia 1994c). About 36% of people who have a heart attack die within 12 months (Martin et al. 1987). The National Health Survey 1989-90 collected information on illness conditions experienced and reasons for action during the two weeks prior to interview. Hypertension was the third most common illness reported during the reference period; only the common cold and headache

were reported more often. Hypertension was also the reason for the most recent doctor consultation for 223,000 people, and heart disease the reason for 66,000 people. Almost 1.7 million people reported taking medication for blood pressure or a heart condition (Australian Bureau of Statistics 1992b).

Costs

Mortality and morbidity from cardiovascular disease place an enormous economic burden on Australians. The total cost of cardiovascular disease was estimated to be $3,390 million in 1989-90. Direct costs of health care, which include hospital, nursing home, medical and pharmaceutical costs, amounted to $2,240 million. Indirect costs, that is those associated with sick leave and premature death, accounted for $1,140 million. The financial impact was greatest for coronary heart disease ($1,090 million), followed by stroke ($640 million) and hypertensive disease ($550 million) (Australian Institute of Health and Welfare 1994, p20). Costs to the community, such as those associated with family members acting as carers, were not included. Nor were the detrimental effects on mental and social health, and quality of life which are difficult to quantify.

2.2 Risk factors for cardiovascular disease

A comprehensive review of the evidence which has established the credentials of each cardiovascular risk factor would be a daunting task and is outside the purpose of this thesis. A brief overview of the evidence is provided for the major risk factors included in the Risk Factor Prevalence Study with particular emphasis on major studies and recent review articles. Results from recent Australian studies are mentioned where they are relevant. Although the focus is on coronary heart disease and stroke, most risk factors are also implicated in the causal mechanism for other cardiovascular diseases. Additional, and sometimes more detailed, discussion of the role of individual risk factors in the aetiology of cardiovascular disease is given in the discussion sections of chapters 4 to 8.

Major risk factors

The cardiovascular risk factors of most preventive importance are cigarette smoking, high blood cholesterol, high blood pressure, obesity and a sedentary lifestyle (Castelli and Leaf 1985; Kirk-Gardner and Crossman 1991; Manson et al. 1992a; Rich-Edwards et al. 1995). Risk factors tend to interact, and the effect of two or more risk factors within the one individual is generally greater than the addition of the individual risks (Manson et al. 1992a). With high incidence populations, most evidence suggests a continuous increasing relationship

between single and combined risk factor levels and the risk of a coronary event rather than the existence of a threshold level of critical importance (Blackburn et al. 1987). Since combined modest risk factor elevations are as strongly predictive of coronary heart disease risk as severe elevations of single risk factors, and such combinations involve large numbers of people in high-risk societies, most excess coronary events come from the central part, rather than the extremes, of risk factor distributions (Blackburn et al. 1987).

Cigarette smoking

A recent Australian study (Chun et al. 1993) showed that, among men aged 35-69, smokers were 2.9 times more likely (relative risk) than non-smokers to suffer a first coronary event (95% CI: 2.7-3.1). The corresponding figure for women was 3.5 (95% CI: 3.2-3.8). Population studies have demonstrated a clear dose-response relationship between smoking and coronary heart disease with up to a five-fold increase in risk of fatal coronary heart disease among heavy smokers (U.S. Department of Health and Human Services 1983; Willett et al. 1987). Passive smoking has been associated with increased risk of heart disease (Steenland 1992), with the excess risk of heart disease for non-smokers living with smokers estimated to be about 30% (Glantz and Parmley 1991). The risk of coronary heart disease is greatly increased when cigarette smoking is combined with other risk factors, particularly hypertension or high blood cholesterol (Chun et al. 1993; U.S. Department of Health and Human Services 1983).

There is also strong evidence of an increased risk of stroke among cigarette smokers (Shaper et al. 1991; Robbins et al. 1994; Wannamethee SG et al. 1995). A meta-analysis of thirty-two studies estimated the overall risk of stroke for cigarette smokers to be 1.5 times that for non-smokers (95% CI: 1.45-1.58), and showed that the excess risk for smokers increased with the number of cigarettes smoked per day (Shinton and Beevers 1989).

Smoking cessation leads to a marked decrease in the risk of cardiac arrest, coronary death, myocardial infarction and stroke (as well as other diseases) (U.S. Department of Health and Human Services 1990; Wannamethee SG et al. 1995). Evidence suggests that the excess risk of coronary heart disease is halved within one year of smoking cessation, although case-control studies have produced differing estimates of the time required for the risk of a coronary event in former smokers to approach that of non-smokers (Negri et al. 1994; Dobson et al. 1991a; U.S. Department of Health and Human Services 1990).

Blood lipids

There is strong scientific evidence linking high blood cholesterol with the development and progression of atherosclerosis and subsequent coronary heart disease (LaRosa et al. 1990; Law et al. 1994a). Low-density lipoprotein cholesterol is positively related to the risk of coronary heart disease, while high-density lipoprotein cholesterol is inversely related to the risk of cardiovascular disease mortality (Expert Panel 1993; NIH Consensus Development Panel on Triglyceride 1993; Pocock et al. 1989; Jacobs et al. 1990; Pekkanen et al. 1990; Manson et al. 1992a). Evidence of a causal association between triglycerides and coronary heart disease is mixed (NIH Consensus Development Panel on Triglyceride 1993; Pocock et al. 1989). Recent results suggest that excessive triglycerides is an additional risk factor in the presence of a high ratio of low-density lipoprotein cholesterol to high-density lipoprotein cholesterol (Assmann and Schulte 1992; Manninen et al. 1992).

Studies have found a strong, continuous and positive association between serum total cholesterol and death from coronary heart disease among middle-aged men (Neaton and Wentworth 1992; Klag et al. 1993; Smith et al. 1992). The finding of a strong positive relationship between serum total cholesterol concentration and coronary heart disease mortality among Chinese men and women in Shanghai, whose cholesterol concentrations are considered normal or low by Western standards, suggests there is no threshold level below which lower levels of serum total cholesterol are not associated with lower risks of coronary heart disease (Chen et al. 1991).

Lowering total and low-density lipoprotein cholesterol levels will reduce the incidence of fatal and non-fatal coronary heart disease (Law et al. 1994b; LaRosa et al. 1990; Yusuf et al. 1988). Results from cohort studies show that a 10% decrease in serum total cholesterol concentration is associated with a 54% decrease in the risk of coronary heart disease for men aged 40 years, and a 20% decrease at ages 70 and 80 (Law et al. 1994b). Results from clinical trials suggest that the full reduction in risk of coronary heart disease is achieved within 5 years of lowering cholesterol (Law et al. 1994b).

There is little evidence of a significant association between serum total cholesterol level and overall stroke mortality (Neaton et al. 1992; Smith et al. 1992; Shaper et al. 1991; Wilhelmsen 1990; Menotti et al. 1990). Data from the Multiple Risk Factor Intervention Trial (Neaton et al. 1992) suggest that this may be because death from non-haemorrhagic stroke is associated with higher cholesterol levels (also reported by Lindenstrøm et al. 1994), while death from intracranial haemorrhage is associated with lower cholesterol levels. A meta-

analysis of randomised controlled trials has indicated that, at least for middle-aged men, lowering serum cholesterol through modified diet or medication may not reduce stroke morbidity and mortality (Atkins et al. 1993).

Hypertension

High blood pressure is a major cardiovascular disease risk factor for both men and women (Joint National Committee 1993; Kannel 1991). The risk of stroke and of coronary heart disease increases as the level of blood pressure increases (Consensus Panel 1994; Whelton 1994; Clausen and Jensen 1992; MacMahon et al. 1990). Systolic blood pressure is a stronger predictor of coronary death than diastolic blood pressure, although both show a strong graded relationship with coronary heart disease mortality (Neaton and Wentworth 1992). Hypertensive people have a two– to four–fold increased risk of cardiovascular disease compared to normotensive people of the same age, depending on the type of cardiovascular disease (Kannel 1991).

A recent Australian population-based case-control study (Al-Roomi et al. 1990) examined the risk of first acute myocardial infarction and first stroke associated with hypertension (defined as being on current treatment). After adjusting for other risk factors, those who had suffered a first infarction were more likely than the controls to be receiving treatment for hypertension (odds ratio 2.6, 95% CI: 1.9-3.4). Men and women who had suffered a first stroke were more likely than controls to have hypertension (OR 3.1; CI 2.0-4.8) (Al-Roomi et al. 1992). Further, those being treated for high blood pressure were more likely to suffer a haemorrhagic stroke (OR 5.5, 95% CI 2.4-12.8) and more likely to suffer a non-haemorrhagic stroke (OR 2.5, 95% CI: 1.5-4.3) than those not being treated for high blood pressure.

Hypertension appears to be an important risk factor for cerebral infarction and intracerebral haemorrhage, however, there is conflicting laboratory and clinical evidence regarding the status of hypertension as risk factor for subarachnoid haemorrhage (Salonen et al. 1982).

Results from prospective observational studies, with a mean follow-up period of 10 years, show that prolonged reductions in usual diastolic blood pressure of 5–10 mmHg were associated with at least 34–56% less stroke (MacMahon et al. 1990). The corresponding reductions in coronary heart disease risk were 21–37%. In randomised drug trials with a mean treatment duration of 5 years and mean reduction in diastolic blood pressure of 5-6 mmHg, both fatal and non-fatal stroke was reduced by 42% (95% CI: 33-50%), even if diastolic blood pressure was below 110 mmHg at the start of treatment (Collins et al. 1990).

The reduction in total coronary heart disease of 14% (95% CI: 4-22%) was less marked than that for stroke.

Obesity

Recent reviews of the health implications of obesity concluded that obesity is an independent risk factor for death from coronary heart disease, and that the risks associated with obesity increase with its severity (Pi-Sunyer 1993a; Pi-Sunyer 1991). These risks include high blood pressure, hypertriglyceridemia, decreased levels of high-density lipoprotein, increased levels of low-density lipoprotein, diabetes mellitus and insulin resistance. Abdominal (central) obesity has been found to be a better predictor of coronary heart disease than overall obesity (Sjöström 1992b; Björntorp 1992; Kannel et al. 1991; Yao et al. 1991; Pi-Sunyer 1993a; Roncari 1992; Bengtsson et al. 1993; Prineas et al. 1993), and to correlate better with other cardiovascular risk factors (Loos and Halais 1991), including psychosocial factors (Björntorp 1988).

Studies have not consistently found a relationship between overall obesity and coronary heart disease, however, methodological differences and limitations may explain some of the inconsistency (Pi-Sunyer 1993a; Manson et al. 1987; Larsson 1992). For example, length of follow-up is an important consideration which varies between studies, and obesity has consistently been demonstrated to be a risk factor for cardiovascular disease morbidity and mortality in studies with follow-up periods greater than 10 years (Pi-Sunyer 1991; Sjöström 1992a; Lee et al. 1993; Rissanen et al. 1991; Shinton et al. 1991). The association between obesity and coronary heart disease may also be underestimated because of failure to control for cigarette smoking, which is strongly associated with lower body weight and is itself a major coronary risk factor (Pekkanen et al. 1994; Manson et al. 1987). There is also debate about whether it is appropriate to control for physiological and metabolic effects of obesity, such as hypertension and hyperglycemia, which are intermediate steps in the causal pathway (Pekkanen et al. 1994; Manson et al. 1987). Studies which do show obesity to be a risk factor for coronary heart disease independently of potential confounders suggest that other mediating factors may be involved (Pekkanen et al. 1994).

There is strong evidence that weight reduction reduces the incidence and severity of cardiovascular disease risk factors, such as non insulin-dependent diabetes mellitus, hypertension, and abnormal lipid and lipoprotein levels, in overweight persons (NIH Technology Assessment Conference Panel 1993; Higgins et al. 1993; Pi-Sunyer 1993b; Benotti et al. 1992). Evidence that weight loss leads to a reduction in cardiovascular disease mortality is inconclusive,

partly because the reason for the weight loss is often unknown and may have been due to pre-existing illness (Williamson and Pamuk 1993; NIH Technology Assessment Conference Panel 1993; Higgins et al. 1993), and partly due to the difficulty of interpreting follow-up data (Brownell and Wadden 1992).

There is some evidence that marked fluctuations in body weight may be associated with an increased risk of heart disease, although there is no evidence that the association is causal (Lissner et al. 1991; Hamm et al. 1989; Garrow 1992; Blair et al. 1993). In animal models, weight cycling is associated with adverse cardiovascular and metabolic changes (Ernsberger and Koletsky 1993).

Physical inactivity

The evidence suggests that physical inactivity is a risk factor for the development of coronary artery disease, that physical inactivity and cardiovascular mortality and risk factors are related, and that moderate physical activity appears to be protective for cardiovascular disease, disability and death. Based on this evidence, expert bodies recommend regular moderate exercise as a beneficial activity to prevent cardiovascular disease (Fletcher et al. 1992; National Heart Foundation of Australia 1991a; National Health and Medical Research Council 1987a; National Health and Medical Research Council 1992a; The Coronary Prevention Group 1992; The Royal College of Physicians 1991).

A recent meta-analysis found that the relative risk of coronary heart disease death for sedentary compared to active occupations was 1.9 (95% CI: 1.6-2.2), while the relative risk of coronary heart disease death for sedentary compared to active groups in non-occupational studies was 1.7 (95% CI: 1.2-2.3) (Berlin and Colditz 1990). It has been suggested that the relative risk of inactivity is similar in magnitude to that of hypertension, hypercholesterolemia and smoking (Powell et al. 1987).

A strong, graded inverse relationship between physical fitness and cardiovascular disease mortality has been reported in many studies (Sandvik et al. 1993; Blair et al. 1989; Ekelund et al. 1988; Slattery and Jacobs 1988; Lakka et al. 1994), and physical fitness has been found to be a better predictor of future coronary heart disease than physical activity (Sobolski et al. 1987). Both physical activity and physical fitness may be protective against hypertension (Blair et al. 1984; Reaven et al. 1991; Paffenbarger et al. 1983; Paffenbarger et al. 1991), and may reduce blood pressure independently of weight loss (Arroll and Beaglehole 1992).

Few studies have examined the relationship between exercise and stroke (Haskell et al. 1992), and most have shown physical inactivity to be associated with an increased risk of stroke (Wannamethee and Shaper 1992). A cohort study of British middle-aged men found that moderate physical activity was associated with a reduced risk of stroke (Wannamethee and Shaper 1992). Physical activity was associated with a reduced risk of stroke among older middle-aged nonsmoking men in the Honolulu Heart Program (Abbott et al. 1994). A beneficial effect of vigorous exercise in early adulthood on the development of stroke in later life has been reported (Shinton and Sagar 1993).

Exercise may increase high-density lipoprotein cholesterol concentration, lower the ratio of low-density to high-density lipoprotein cholesterol, lower plasma triglycerides, decrease insulin production, increase insulin sensitivity, and reduce the risk of abdominal obesity. The beneficial effects depend on the frequency, duration and intensity of the exercise and some may only be short-lived effects that are lost after exercise training stops (Haskell et al. 1992; Blair et al. 1992; Blair 1993; Wood et al. 1988). Exercise training may have beneficial effects for coagulation and fibrinolysis (Haskell et al. 1992) and the risk of developing non-insulin dependent diabetes mellitus (Helmrich et al. 1991; Manson et al. 1991a; Manson et al. 1992b). Recent evidence shows that physical activity does not have to be of vigorous intensity to have a favourable impact on cardiovascular health. Moderately intense leisure time activity also provides health benefits. (Blair et al. 1992; Shaper and Wannamethee 1991; Blair et al. 1989; Leon et al. 1987)

Although moderate physical activity is generally beneficial, there is evidence of a temporarily increased risk of a sudden cardiac event during bouts of acute physical activity (Kohl et al. 1992; Willich et al. 1993; Mittleman et al. 1993). This risk is substantially lower among persons who undertake regular physical activity compared to those who are habitually sedentary.

Alcohol

Heavy alcohol consumption is associated with increased cardiovascular disease morbidity and mortality in both men and women (Kannel 1988; Hanna et al. 1992; National Heart Foundation of Australia 1991b). Excessive alcohol use is also associated with an increased risk of coronary heart disease (Kannel 1988) and stroke, particularly subarachnoid haemorrhagic stroke (Donahue et al. 1986; Gill et al. 1986; Stampfer et al. 1988; Shaper et al. 1991).

Despite the increased risk of cardiovascular disease associated with excess alcohol intake, there is a wealth of evidence suggesting that light to moderate

alcohol consumption reduces the risk of coronary heart disease (Cullen et al. 1993; Jackson et al. 1991; Jackson et al. 1992; Rimm et al. 1991; Klatsky et al. 1992; Friedman and Klatsky 1993). In particular, light to moderate consumption of alcohol is associated with an estimated 25 to 45% reduction in the risk of myocardial infarction compared to non-drinkers (Manson et al. 1992a).

The reasons for this apparently beneficial effect of modest alcohol intake are not clear. Alcohol consumption is associated with high blood pressure (Beilin and Puddey 1992; Regan 1990; Russell et al. 1991; Wakabayashi et al. 1994; Ueshima et al. 1992; Witteman et al. 1990), which could be expected to increase rather than decrease risk but this association may not be relevant at low levels of alcohol intake. Alcohol consumption is positively associated with high-density cholesterol levels, which may confer a protective effect.

It has been suggested that the apparent protective effect is an artefact of drinkers at high risk of coronary heart disease becoming non-drinkers thereby increasing the risk of the non-drinking group (Shaper 1990; Kaufman et al. 1985). The findings of a large case-control study refuted this argument (Jackson et al. 1991) and it gained no support from a comprehensive review of the evidence (Marmot and Brunner 1991). A recent study, however, found weak support for the hypothesis (Shaper et al. 1994).

Other risk factors

Habitual diet[1] has been linked to risk factors such as blood lipids, hypertension and obesity, and to coronary heart disease (Shrapnel 1992). Diabetes mellitus and oral contraceptive use among pre-menopausal women have also been associated with increased risk of cardiovascular disease (Sniderman 1992; Manson et al. 1991b; Stehbens 1990; Milne and Vessey 1992). There is evidence that low-dose aspirin prophylaxis (Manson et al. 1992a; Hennekens et al. 1988) and oestrogen replacement therapy in post-menopausal women (Manson et al. 1992a; Stampfer and Colditz 1991) may reduce the risk of coronary heart disease. Early health disorders such as high blood pressure, and lifestyle behaviours such as smoking, have increasingly been seen as a product of social and economic conditions and opportunities (Marmot and Morris 1984; McMichael 1989). For this reason, socioeconomic factors, such as poverty and unemployment, are treated often as risk factors. Also, demographic characteristics such as age, sex, race and family history are viewed as risk factors sometimes, although they are obviously not risk factors which can be

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[1] Dietary factors include saturated fatty acids, the n-6 polyunsaturated fatty acids, the n-3 polyunsaturated fatty acids (fish oils), mono-unsaturated fatty acids, trans fatty acids, total fat intake, dietary cholesterol, sodium and potassium, as well as alcohol.

modified. Genetic components are involved directly in the aetiology of cardiovascular disease and through biomedical and behavioural risk factor pathways (Oberman et al. 1994; Wilson 1993, Stehbens 1990; Holtzman 1989).

2.3 The importance of monitoring trends in risk factors

Beaglehole (1990) has argued that the decline in coronary heart disease mortality is the net effect of changes in incidence and case fatality which flow from changes in population risk factors and medical care. The relative importance of trends in risk factors and medical care of coronary events to the decline in coronary mortality can be examined by considering trends in incidence and case fatality. A decline in incidence suggests reduced levels of coronary risk factors in the population. A decline in case fatality while incidence remains constant suggests more effective medical intervention in acute coronary events or less severe disease, perhaps associated with lower risk factors (Leeder et al. 1984). Evidence of trends in incidence and case fatality in Australia has been provided by the WHO MONICA[1] centres in Perth and Newcastle by linking population-based disease registers, hospital discharge data and mortality records.

In 1984, Martin and colleagues reported that case fatality at 28 days and up to one year after admission to hospital for myocardial infarction (acute or sub-acute) remained constant in Perth between 1971 and 1979. They concluded that improved survival after hospital admission did not contribute to the fall in deaths during this period. In 1989, Martin et al. demonstrated marked falls in age-adjusted incidence rates for myocardial infarction in Perth, Western Australia, between 1971 and 1982, and also showed that over 80% of the decline in mortality was in deaths outside hospital. The authors concluded that the decline in the incidence of myocardial infarction contributed substantially to the observed decrease in coronary heart disease mortality, and that the most plausible explanation for the decline in incidence was a reduction in the prevalence of risk factors for myocardial infarction. More recently, Hockey et al. (1991) reported that case fatality for men admitted to hospital for acute myocardial infarction improved from the mid 1970s to the mid 1980s, while for women there was no improvement. Results from Newcastle are similar to those for Perth. In 1988, Dobson et al. reported significant declines in the incidence of acute myocardial infarction in the Hunter Region of New South Wales from 1979 to 1985 and no decline in case fatality rates. Recent reports indicate that

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[1] World Health Organization international project (*Moni*toring trends and determinants in *ca*rdiovascular disease).

case fatality has fallen in this region since 1985 (Dobson et al. 1991b; Heller et al. 1992).

Results from Perth and Newcastle suggest, therefore, that the fall in mortality from coronary heart disease was initially due to falling incidence but that, more recently, improved survival after a heart attack has contributed to the decline for men. There is supportive evidence for a recent decline in case fatality. Trends have been documented in the pharmacological management of myocardial infarction in the Perth MONICA project which would partly explain observed improvements in case fatality (Thompson et al. 1992). Also, the results of a community-based cross-sectional survey of patients living in the Perth Statistical Division who were admitted to hospital with acute myocardial infarction during 1984-88 suggest that follow-up care after acute myocardial infarction was both comprehensive and widespread (Czarn et al. 1992). It has also been demonstrated for the Newcastle MONICA project that large changes in drug treatment of patients with acute myocardial infarction occurred in the second half of the 1980s (Heller et al. 1992). National statistics indicate an increase in prescribing of anti-hypertensive drugs during the 1980s (Hurley et al. 1990) and a dramatic increase in the use of cholesterol lowering drugs towards the end of the decade (Henry et al. 1991). The steady rise in coronary artery bypass surgery (National Heart Foundation of Australia 1994b) and the introduction of coronary angioplasty (National Heart Foundation of Australia 1994a) during the 1980s would also be expected to improve case fatality.

Other research relevant to the role played by risk factors includes the ecological analysis in 1980 by Dwyer and Hetzel which compared coronary heart disease mortality in Australia, the United States and the United Kingdom with changes in risk factors. The authors concluded that the substantial decline in the United States and Australia correlated to some extent with life style changes, particularly in relation to diet (increase in vegetable fat consumption and a decrease in animal fat) and smoking. Only a limited number of risk factors were considered, however, and modelling was univariate and did not take into account interaction between the risk factors.

Dwyer and Hetzel (1980) proposed a correlation between the decline in coronary heart disease mortality and dietary changes based on five foods known to be related to plasma cholesterol concentration. Dietary changes were said to be the substitution of dietary saturated fat by polyunsaturated fat through increased use of vegetable oils and margarine and decreased use of animal fats. The food data used were from the national apparent consumption series. Conclusions about the role of polyunsaturated fats were predicated on

assumptions that 'table margarine' approximated 'polyunsaturated margarine' and that 'vegetable fat' approximated unsaturated fat (Dwyer and Hetzel 1980, Hetzel et al 1989). However, the limitations of the apparent consumption fat data make it inadequate to support these assumptions, since the vegetable oil data are estimates and fatty acid data are not available (Lester 1994). In fact, a high proportion of the vegetable oil supply was coconut oil (a saturated oil) and most margarine was highly saturated until the late 1970s (National Health and Medical Research Council 1992b). Thus the role of dietary fat in the decline in coronary heart disease mortality since the late 1960s in Australia is yet to be resolved.

Demographic analyses of mortality data for period and cohort effects are also relevant. Dobson et al. (1981) demonstrated that a cohort effect operated from 1950 to 1966, suggesting that the increase in coronary heart disease during this period was due to increased exposure of successive generations to coronary risk factors. In contrast, during the late 1960s, the trend in coronary heart disease mortality rate changed direction in most age groups and both sexes simultaneously. This is consistent with a decreased effect, throughout the whole community, of risk factors which are causally related to coronary heart disease, and with improved medical management of patients with coronary heart disease. In 1989, Al-Roomi et al. demonstrated that the period effect had been dominant since the late 1960s, and that no cohort effect was apparent for either coronary heart disease or stroke. The authors noted that changes in diet, cigarette smoking, treatment of hypertension and coronary care were consistent with the mortality trends.

The contribution of risk factors to the decline in cardiovascular disease in Australia has been estimated at 50% for men and 75% for women (Dobson 1987). These estimates are of the same order of magnitude as those obtained earlier for the United States (Goldman and Cook 1984). The Australian figures were calculated by applying parameters derived from the Framingham Heart Study[1] to the observed Australian changes in blood pressure, cholesterol and smoking during the 1970s and 1980s. This approach has numerous methodological problems (Blackburn 1989; Sprafka et al. 1990), and it is questionable whether parameters derived from other populations at other times can be justifiably applied to Australia during the 1980s (Chambless et al. 1990). The model fails to take into account changes in other risk factors, trends in multiple risk factors, and the lag time between risk factor modification and subsequent effect on cardiovascular risk which is known to vary between risk

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[1] Recruitment to the Framingham Heart Study commenced in 1948 and surveillance has continued since.

factors (Manson et al. 1992a). Although estimates of the contributions of risk factor (and medical care) changes to mortality trends may be improved, some methodological problems may not be soluble (Blackburn 1989). Nevertheless, after summarising papers presented at an international workshop on trends and determinants of coronary heart disease mortality, Blackburn concluded with advice which is strongly supportive of monitoring trends in risk factors:

> 'With the perpetual uncertainty of conclusions about direct causes of mortality trends, a focus should remain clear: risk factors are demonstrably the causes of cardiovascular disease; mass elevations of risk factors are the causes of mass disease; most humans exhibit risk when exposed to unfavourable environments; and change in population levels of risk factors is required for an effective preventive strategy.' (Blackburn 1989)

Overall, the evidence clearly suggests that improvements in Australia's risk factor profiles have played a substantial part in the decline in coronary mortality, even though their relative contribution to the mortality trend is difficult to estimate with confidence. It is also clear that the monitoring of risk factor trends is an important element in the rational explanation of time trends in mortality. Even so, the lack of appropriate data in Australia has meant that little has been published on Australian trends in the major risk factors for coronary heart disease.

In 1987, Dobson reviewed the range of published data on cardiovascular risk factors among adults in Australia from 1966 to 1983 and found that smoking decreased among men but increased among younger women, while mean cholesterol and systolic blood pressure levels decreased among men and women. Diastolic blood pressure showed no significant or consistent changes among men but some decrease among women. Inferences from the analysis were limited by major variations in methods between the various studies, for example, in geographical coverage and in representativeness. In particular, cholesterol trend estimates were heavily dependent upon high levels observed in the 1966 Busselton survey, a township in Western Australia (Thompson et al. 1988).

In 1989, Jamrozik and Hockey compared the 1980 and 1983 Risk Factor Prevalence Surveys and detected beneficial trends in smoking and blood pressure. Total cholesterol levels did not change, and there were some desirable changes in dietary behaviour and physical activity, but the prevalence of people overweight or obese increased. Their analysis did not allow for independent sex or age group trends, and changes to the survey questionnaire

may have confounded some trends, especially those for dietary factors and exercise.

Other trend data relate to single risk factors. In 1990, Bauman et al. examined trends in exercise participation using data from five national surveys conducted during the period from 1984 to 1987, They found a significant decrease in the proportion of adults reporting to be totally sedentary and a slight increase in those who participated in regular physical activity. An important time series for smoking prevalence is that reported by Hill et al. (1991) which shows declines in the prevalence of smoking for men and women since the mid 1970s, but a greater decline among men.

In conclusion, although the monitoring of trends in risk factors provides essential information for understanding trends in coronary mortality, information of trends in risk factors in Australia has been piecemeal and limited. The results in chapter 4 represent an important addition to the risk factor trend data available in Australia because of the wide range of risk factors considered. The physical examination component of the Risk Factor Prevalence Study provide nation-wide data on trends in biomedical risk factors which have not previously been available in Australia and are not commonly available in other countries. The analysis extends the previous comparison of the 1980 and 1983 surveys (Jamrozik and Hockey 1989) to a nine year period and gives results separately for men and women. Because age adjustment or age standardisation can mask age specific trends, results are also given for 10-year age groups. The chapter identifies changes in levels of risk factors which might be associated with the continuing fall in cardiovascular mortality and those risk factors which have an unfavourable trend.

2.4 Blood pressure measurement error

Basic monitoring procedures had detected marked variation in measurement technique for blood pressure between centres and between surveys, and, since blood pressure is a major risk factor for cardiovascular disease, it was important to evaluate the effect of this variation on estimates of trends. The mercury sphygmomanometer is commonly used in epidemiological studies and was the preferred method of measuring blood pressure in the Risk Factor Prevalence Study. Both the American Heart Association and the British Hypertension Society agree that, with careful attention to detail, the mercury sphygmomanometer provides accurate, reliable and reproducible blood pressure measurements (Stewart and Padfield 1992). Experience has shown, however, that this is not readily accomplished in epidemiological studies which have multiple examination centres and observers (Bruce et al. 1988; Hense et al.

1990). Despite methodological improvements and sophisticated training methods to standardise measuring techniques between observers, the few data that have been published on observer variation in blood pressure indicate that it has proven notoriously difficult to control (Choi et al. 1978; Eilersten and Humerfelt 1968; Hypertension Detection and Follow-up Program Cooperative Group 1978; Dischinger and DuChene 1986; National Center for Health Statistics et al. 1986; Bruce et al. 1988; Hense et al. 1990).

The first recorded systematic investigation of observer error, conducted by Wilcox in 1960, demonstrated considerable within and between observer variability in the measurement of blood pressure by nurses. The author concluded that new and improved methods of training were required, and expressed the opinion that physicians would perform more reliably. In 1964, Rose et al. demonstrated inter-observer error of the same magnitude among physicians as Wilcox had observed among nurses, and noted three forms of observer error: systematic error; terminal digit preference; and prejudice for, or against, certain pressure values. The following year, Rose (1965) described a training technique for standardisation of methods used by blood pressure observers. In 1966, Armitage et al. demonstrated a fall of 3.6 mmHg in systolic pressure between duplicate readings when the second was made immediately after the first. It was noted that the prevalence of hypertension would have been significantly overestimated by single readings and concluded that the precision and sensitivity of studies of blood pressure may be materially improved if each subject is examined more than once.

Little attention has been given to the effect of these phenomena in epidemiological studies. Hessel (1986) examined the effect of terminal digit preference on epidemiological analyses by analysing blood pressure measurements taken by 12 medical officers during a routine industrial screening program. He concluded that digit preferencing would have the effect of reducing the power of statistical tests, thereby making it more difficult to assess associations between blood pressure and other potential risk factors.

The most elucidating study is the analysis of blood pressure measurements for 51 different populations which had participated in baseline surveys for the WHO MONICA Project (Hense et al 1990). The authors applied last digit preference scores and proportions of identical duplicate measurements from the MONICA surveys to fictitious blood pressure distributions and demonstrated that the former affected predominantly the shape of the distribution, whereas the latter may cause a shift in the entire blood pressure distribution. When making repeat measurements, the proportion of identical

duplicate measurements is an indicator of a tendency to adjust the second reading to make it conform with the first reading. Its effect is to moderate the magnitude of the average observed fall between repeat readings (Armitage et al. 1966). This phenomenon has recently been suggested as a contributing factor to why the Hawksley random zero sphygmomanometer produces lower mean values and greater intra-observer variability than the standard mercury sphygmomanometer (Parker et al. 1988; Canner et al. 1991; Hense 1991).

In conclusion, the evidence suggests that variation in blood pressure measurement technique using normal mercury sphygmomanometers can affect estimates of trends in epidemiological studies but the magnitude of this effect is unknown. Blood pressure measurement in the Risk Factor Prevalence Study consisted of two consecutive readings recorded to the nearest 2 mmHg, consistent with the MONICA protocol. These data provided the opportunity to demonstrate the variation in measurement technique which occurs between specially trained nursing sisters in a survey setting and to estimate the magnitude of the effects described by Hense et al (1990). Observer variation is used to estimate the effect of last digit preference for zero on prevalence estimates of high blood pressure, and the effect of identical duplicate measurements on comparative cross-sectional or longitudinal analyses of mean pressures. The findings, given in chapter 5, are applicable to other studies which follow the MONICA protocol for measuring blood pressure, and were used to assess the trends in blood pressure reported in chapter 4.

2.5 Cardiovascular differentials among immigrants

After two decades of multiculturalism, Australia has one of the most culturally diverse societies in the world. Around 23% of people living in Australia were born overseas, which represents a significant proportion of the Australian population. Immigrants are not a homogeneous group, originating from more than 100 different countries, most representing a relatively small proportion of the total immigrant population (Young 1992c). The composition of the migration stream has changed over time. Before World War II, migration to Australia was almost entirely from the British Isles. During the post-war period, over 60% of immigrants came from countries other than Britain. They arrived in Australia in distinct waves. Eastern European refugees were followed by immigrants from Western Europe, who were later replaced by immigrants from the Mediterranean basin and finally by Asians. The latter initially came from the Indian subcontinent, afterwards from Lebanon and most recently from Indo-China (Krupinski 1984; Jupp 1990). As a result, there is wide variation between immigrant groups with respect to age structure and

duration of residence (Young 1992c). It is important that these differences in demographic characteristics are recognised in epidemiological research.

The increasing cultural diversity of the Australian population has changed the food habits and lifestyle of Australians, while in turn, the availability of foods, changed economic circumstances and the Australian culture affects the eating habits and social patterns of immigrants. This has implications for the risk factor, health and mortality profiles of the Australian people. The study of the pattern of disease, in particular cardiovascular disease, among immigrant groups and the Australian-born within the community informs the planning and development of appropriate health initiatives and contributes to the knowledge of factors which influence differentials in disease risk.

Cardiovascular mortality

Most, but not all, of Australia's immigrant groups continue to experience lower mortality rates from coronary heart disease than the Australian born. The first published analysis of differential mortality from cardiovascular disease in immigrants to Australia reported lower death rates during 1962-66 among immigrants from England and Wales and Italy than the Australian-born and Scottish immigrants (Stenhouse and McCall 1970). The death rates of the former were greatest among those who had been resident in Australia the longest and exceeded that of their country of origin. Numbers of deaths were not given, however, and are likely to have been very small in some cells.

In 1977, Powles and Birrell produced standardised mortality ratios for overseas and Australian-born, for each local government area in Victoria, for 1969-73, and demonstrated that immigrants had lower rates for coronary heart disease, hypertension and stroke. Their report contained data supplied by Stenhouse and McCall showing age-specific (total) death rates were lower among Italian and Greek male immigrants than in their county of origin, which were in turn lower than those for the Australian-born. Also, among male Greek immigrants, death rates were higher among those who had been resident in Australia for longer. The authors suggested a major role for pre-migratory environments and selective migration, and a lesser role for the influence of a genetic advantage, in the lower mortality among immigrants. Mortality from coronary heart disease was markedly lower in local government areas with a high proportion of immigrants from Mediterranean countries, even though those same areas were relatively deprived in terms of income, job status and housing quality.

Lower mortality among immigrants from Italy and Greece was also reported by Najman in 1978. Differential mortality during 1965-67 among eight birthplace

groups was reported for a range of causes of death, including coronary heart disease. The analysis by period of residence related to all immigrants and may have been confounded by the changing distribution of Australia's immigrant intake over time.

A systematic analysis by Dasvarma (1980) produced standardised mortality ratios for 1970-72, for eleven birthplace groups, and for diseases including coronary heart disease and stroke. Almost all immigrant groups experienced lower mortality from coronary heart disease than the Australian-born population. Exceptions were Polish immigrants, German male immigrants and females from New Zealand and North America. Death rates tended to increase with increasing period of residence (a dichotomous variable defined at 10 years), but remained lower than levels among the Australian-born. The lack of information on the diet and lifestyles of immigrant groups was noted.

In 1982, Dunt demonstrated markedly low mortality from coronary heart disease and stroke among Southern Europeans during 1970-72, attributed in part to medical screening of potential immigrants and low rates in the countries of origin. Between 1966 and 1976, death rates for stroke fell by 40% in Southern European men and women. Southern European women also experienced a fall (27%) in coronary heart disease mortality but not so Southern European men (+4%), in contrast to Australasian men (-18%). The analysis included a comparison of trends in mortality rates between Southern European immigrants and their countries of origin for all causes and coronary heart disease. The conclusion that trends were similar was not based on strong evidence.

In 1984, mortality from coronary heart disease for the period 1969-1978 was shown to be lower among immigrants, with death rates to those born in Greece, Italy and Yugoslavia about half the average rate. Falls in mortality were greatest among the Australia-born, who also had the highest initial rates, and Southern European females (Gibberd et al. 1984). Results from two regional studies also demonstrated mortality differentials for cardiovascular disease. Male Chinese in New South Wales during 1969-78 were more likely to die of cardiovascular disease than the Australian population while female Chinese immigrants were less likely (Zhang et al. 1984). Mortality from stroke was higher than expected among Chinese male immigrants; mortality from coronary heart disease lower than expected among Chinese female immigrants. Others showed that death rates for coronary heart disease were higher among Maltese immigrants in Victoria during 1978-79, and lower among Italian-born immigrants, than rates for all Victoria (Martin et al. 1984).

A series of publications by Young (1986a; 1986b; 1987; 1991; 1992b) represent a substantial contribution to the literature on the mortality experience of immigrants to Australia. The analyses systematically considered total mortality, a wide range of causes of death, a large number of birthplaces, and period of residence. Mortality from coronary heart disease varied markedly between birthplaces with most countries experiencing lower mortality than the Australian-born. Standardised mortality ratios for coronary heart disease for men aged 15-74 varied from 52 (Italy) to 131 (South Asia), and from 48 (Greece) to 138 (Poland) for women. These differentials were attributed to variation in selection criteria (official selection procedures and self-selection), differences in diet and lifestyle, the level of mortality in the country of origin, and genetic predisposition to disease. A comparison of the data for the periods 1980-82, 1984-86 and 1987-89 showed consistently low mortality from cardiovascular disease for several of the major immigrant groups in Australia, notably those from Italy, Yugoslavia, Greece, the Netherlands, Vietnam, Lebanon (males), and Central and South America (Young 1992b). Cardiovascular mortality was consistently higher among Poles, Maltese females and Pacific Islanders than the non-immigrant population.

Generally, mortality rates from cardiovascular diseases were higher among those with a longer duration of residence. For many birthplaces, however, mortality rates remained much lower than the Australian-born rates even after more than 15 years in Australia, and some showed very little change (Young 1986b). Young pointed out that the data refer to the experience of different arrival cohorts, and therefore differences according to period of residence could reflect different waves of immigrants from a given birthplace. Increases in mortality which occur with increasing duration of residence could reflect a convergence to the underlying level of mortality in the country of origin or to the overall Australian experience.

In addition, wide variation was demonstrated in mortality rates from causes other than cardiovascular diseases. Each immigrant group experienced a different mortality profile, most likely related to the cause of death profile in the country of origin, diet, lifestyle and interaction with the Australian environment (Young 1992a). Powles and Gifford (1990) suggest that diet and lifestyle overwhelm other influences in the incidence rates of non-communicable diseases and are important influences on mortality differentials.

Risk factors

Although most immigrant groups have lower, and in many cases significantly lower, cardiovascular mortality than the total Australian population, there has

been no comprehensive study of the level of risk factors in Australia's varied immigrant population, and whether risk factor profiles are consistent with cardiovascular mortality experience. In particular, the few studies of differentials in biomedical risk factors among immigrant groups have tended to focus on specific immigrant groups or specific risk factors.

Lovell and Prineas, in 1974, reported lower blood pressure levels, less hypertension but greater obesity among middle-aged Italian immigrants in Melbourne compared with their Australian-born counterparts. The following year, Ulman and Abernethy (1975), using data from one of the Melbourne centres of the Australian National Blood Pressure Study, also reported that Italians aged 30-69 years had lower systolic and diastolic blood pressure than Australian-born of the same age and sex. Duration of residence had a small but statistically significant association with blood pressure.

In 1977, a range of biomedical and behavioural risk factors were collected on a sample of Italian immigrants and a sample of Australians of Australian, British or New Zealand parentage, resident in designated areas of Perth, and matched for age and sex (Armstrong et al. 1983). The researchers concluded that the differences found in coronary risk factors were insufficient to explain the markedly lower coronary heart disease mortality rates among the Italian-born. Only lower blood pressure levels among the Italian migrants, also observed by Lovell and Prineas (1974) and Ulman and Abernethy (1975), were consistent with lower mortality rates. No differences were observed in total cholesterol levels. Risk factor levels were influenced little by duration of residence in Australia. As the authors pointed out, neither the Italian migrants nor the Australian-born sample were representative of their respective total populations in Perth, and the samples also differed demographically from each other. The samples were not large enough to allow for statistical adjustment of these variables in the analysis.

Analysis of the 1980 Risk Factor Prevalence Survey demonstrated that the prevalence of overweight or obese men and women was markedly higher among Southern European immigrants (English and Bennett 1985) consistent with previous reports of greater body fatness among Italian immigrants (Lovell and Prineas 1974; Armstrong et al. 1983). Prevalence was lower among Asian immigrants, with the Australian-born in between. Similar differentials were reported based on analysis of self-reported height and weight data collected in the 1989/90 National Health Survey (Mathers 1994b). Reasons for the higher prevalence of excess weight for height among Southern European immigrants are not clear. Genetic predisposition or a cultural preference for greater body

weight may be involved (Webb and Manderson 1990) as might environmental factors following migration to Australia. Powles and colleagues observed a higher mean body mass index among Greek males who migrated to Australia from the island of Levkada than among their siblings and family members who did not, suggesting a tendency for immigrants to increase their weight (unpublished work cited in Webb and Manderson 1990). Certainly, a greater proportion of migrants (60%) felt they had to watch their weight than those who remained on Levkada (21%) (Powles et al. 1988). Socioeconomic status may also be a contributing factor given the inverse association between socioeconomic status and body fatness and the fact that Southern European immigrants to Australia are more likely to have lower educational attainment, lower status occupations, and lower income than the Australian-born population (Young 1992c).

Studies of immigrants' food habits and dietary intakes in Australia have tended to focus on Greek, Italian or Vietnamese communities, and have been bedevilled by unrepresentative samples or samples of insufficient size for adequate statistical analysis. For example, in 1980, Hopkins et al. reported results from the dietary change component of the 1977 Perth study which indicated that Italians migrating to Australia had tended to increase the amount of meat and animal fat in their diet and to decrease their intake of fruit, vegetables and starches, suggesting a change from their native diet toward that of Australians, and potentially undesirable consequences for their experience of chronic disease. However, the samples differed with respect to important demographic characteristics, and sample sizes (170 in each group) were insufficient to permit adequate statistical adjustment. In 1981, Margetts et al. reported the results of the 24-hour dietary record sub-sample and found that Italian men ate less animal fat and more vegetable fat than Australian men; Italian women ate more vegetable fat than Australian women. However, sample numbers were small (77 Italians and 85 Australians) and subject to the same limitations as the previous analysis. Thus, while these analyses provide some evidence that dietary factors may contribute to the beneficial mortality experience of Italian immigrants, generalisation from the study samples is problematic.

Very few dietary studies have been able to compare dietary intake data between the major immigrant groups in Australia. Powles et al. (1990) used 1984 household expenditure data to compare health-related consumption expenditure between five immigrants groups and the Australian-born. The data demonstrated a tendency for low mortality groups (such as immigrants from Italy and Greece) to spend more on fruits, vegetables, cereal products and

fish, and substantially less on alcohol than the Australian-born. However, this was combined with substantial expenditures on red meat and tobacco (Greek males).

The most extensive data are provided by the 1983 National Dietary Survey of Adults (Cashel et al. 1986; English et al. 1987), the first population-based nationwide survey of dietary intakes since 1944. The survey[1] collected 24-hour dietary recalls on 6255 urban-living adult Australians aged 25-64 of whom 1755 were born overseas. It was found that immigrants from Southern Europe and Asia consumed more bread and cereals, particularly white bread, pasta and rice (Asia), more tomatoes and tomato products, leafy greens, and citrus fruits and juices than the Australian-born, but less potatoes and other root vegetables. Total meat consumption varied little between immigrant groups, although the type of meat eaten was different. Southern Europeans and Asians tend to use less dairy products, and butter or margarine consumption was half that of the Australian-born. The contribution of fat to total energy intake was comparatively low among Southern European men and Asian men and women. Immigrants from Southern Europe and Asia consumed higher ratios of polyunsaturated and mono-unsaturated fatty acids to saturated fatty acid intake.

In 1990, Webb and Manderson reviewed the Australian literature on food habits and dietary intake and noted that many studies had substantial methodological deficiencies. They concluded that the existing literature offers few adequate descriptions of food patterns and nutritional status of Australia's immigrants, and there was little conclusive evidence about the nature, extent and timing of changes in diet or nutritional status associated with migration.

The best source of data on behavioural risk factors (smoking, lack of exercise and alcohol intake) is the 1989/90 National Health Survey, a population-based survey by the Australian Bureau of Statistics of over 22,000 households throughout Australia. However, published data are population estimates of absolute prevalence by birthplace, unadjusted for the different age (and sometimes sex) structures of immigrant groups (Australian Bureau of Statistics 1994a). Age standardised ratios by a detailed classification of birthplace have been published by Young (1992b).

This lack of a comprehensive immigrant study of cardiovascular risk factors to date reflects, in part, the difficulties of obtaining population samples that are

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[1] The dietary survey was 'piggy-backed' on the 1983 Risk Factor Prevalence Survey as a large sub-sample.

sufficiently large and representative of the immigrant population. The large number of countries of origin represented in Australia, the relatively low numbers in each immigrant group and their geographical distribution present significant practical difficulties. Added to this are issues related to the collection of biomedical measurements in large-scale probability samples. Data on differentials in biomedical risk factors among immigrant groups are particularly sparse. The National Heart Foundation's Risk Factor Prevalence Study, comprising probability surveys in 1980, 1983 and 1989, represented a unique opportunity for a comprehensive analysis of risk factor differentials for a relatively detailed classification of the immigrant population. By combining the three surveys, a wide range of biomedical and behavioural risk factor data were available on over 6,000 immigrants, representing the major urban areas in Australia. The systematic comparison of risk factors among immigrant groups and their change with duration of residence forms the basis of chapter 6. The chapter assesses whether differences in cardiovascular mortality are related to differences in the risk factor profiles of the various immigrant groups, and whether immigrants' risk factor levels approximate those of the Australian-born with increasing length of stay in Australia.

2.6 Socioeconomic inequalities in cardiovascular disease

The 1980s saw a substantial increase in the literature relating to socioeconomic status and health, comparing the mortality and morbidity experiences of socioeconomic groups within and between countries, documenting the magnitude and trends in inequalities, and exploring explanations for differential health outcomes. A selective overview of this literature is given in the next two sections, sufficient to provide a context for chapters 7 and 8 of the thesis which examine socioeconomic inequalities in cardiovascular mortality and risk factors for Australia and their variation over time. As well as covering Australian activities, the review includes research from Britain and the United States where the association between socioeconomic disadvantage and health status has been the subject of much research and scientific and political debate. Although the discussion focuses on evidence of mortality differentials for cardiovascular disease (or coronary heart disease), evidence for total mortality is included since cardiovascular disease is the major cause of death in the countries reviewed and patterns for total mortality, to a large extent, reflect patterns in cardiovascular disease. But first the meanings of social class and socioeconomic status are discussed.

In Britain, it is common to use the term 'social class' when in the United States 'socioeconomic status' would be used (Marmot 1989), and in Australia both

terms are encountered. In sociological terms, social class and socioeconomic status refer to two distinct stratification systems (Robinson and Kelley 1979). Marx conceptualised social class in terms of the conflict or competition between the upper class (owners of capital) and the lower class (workers) based on the production system. According to Weber, however, social class refers to groups of people with common economic life chances determined by market forces (Quine and Lancaster 1989). Weber accords far greater importance to the marketplace rather than the point of production in the determination of class position. For Weber, class relations are market relations: the mass of non-property owners will have very different class situations depending on skills and credentials they can offer to prospective employers in the market place (Baxter et al. 1991). Socioeconomic status on the other hand is a consensus term which refers to a range of social and economic differences among people based largely on educational and occupational achievements. It involves the hierarchical ordering of people according to prestige and lifestyle. Socioeconomic status is often used to refer to an index, usually a composite score based on occupation, education and income. Some social scientists and most epidemiologists use the terms 'social class' and socioeconomic status' interchangeably (Quine and Lancaster 1989).

Social class is a multifaceted, complex variable which is difficult to conceptualise and measure (Quine and Lancaster 1989; Turrell et al. 1994). Neither Marx nor Weber provided detailed empirical pictures of class structures based on their respective 'models' of class. This has been undertaken by contemporary researchers, notably Erik Wright who constructed a Marxist relational class model[1] and John Goldthorpe who developed a Weberian schema[2]. A crucial task for class theorists has been the emergence of 'new' middle classes - those wage earners who do not own productive property yet whose work situation clearly invalidates their designation as simply manual or 'proletarian' workers (Baxter et al. 1991).

Most measures of social stratification accord with Weber's view that it has three separate but related domains: class, status and power (Liberatos et al. 1988). Class is assumed to have an economic base and implies ownership and control of resources as indicated by measures of income. Status is considered to be prestige or honour in the community and implies access to life chances based on social and cultural factors such as family background, lifestyle and

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[1] Bourgeoisie; Small employers; *Petit bourgeoisie*; Managers; Adviser managers; Supervisors; Semi-autonomous employees; Proletariat.

[2] Upper service; Lower service; Routine non-manual; Small proprietors, own account workers; Lower grade technical, non-manual supervisory; Skilled manual; Semi- and unskilled manual.

social networks. Power is related to a political context. The three indicators that have been used most often to reflect these concepts are occupation, education and income. Occupational classifications based on educational requirements and income (the socioeconomic approach) fall into Weber's class domain. Those ranked by prestige fall into Weber's status domain. Because educational attainment influences lifestyle and social networks it falls into Weber's status domain. However, because it also provides the qualifications to acquire certain occupations and income, education may also be considered to fall into the class domain. Income, the third indicator, falls into Weber's class domain. The three domains are related. For example, differentials in income may influence opportunities for education and provide access to different lifestyles, prestige or power. Thus social class incorporates economic, political and cultural dimensions, each of which may impact on health (Liberatos et al. 1988).

A proper sociological task is to get behind the labels created by any classificatory system and to explore their meaning in the everyday lives and life chances of those on whom the labels are imposed (Macintyre 1986). The concept of social class is viewed by social scientists as crucial to the analysis of society and human behaviour because members of a particular social class are believed to hold similar values, follow similar lifestyles and exhibit similar behaviours. Furthermore, an individual's membership of a social class indicates the probability of enjoying certain benefits, such as good housing and sanitation, sound nutrition and health, substantial education and knowledge, financial security, and non-hazardous and well remunerated employment (Quine and Lancaster 1989). Social class analysis has the potential for examining the way that the organisation of society affects health and disease (Marmot et al. 1987).

Australian society has a clearly definable class structure (Baxter et al. 1991). Researchers in Australia have used measures based on occupation, income, and educational attainment although occupational classifications or occupational prestige rankings have been more common. In the current context, educational attainment indicates knowledge and receptiveness to health education messages, occupation indicates exposure to particular physical or psychosocial factors, and income indicates the power to purchase goods and services relevant to health. Measures based on composite scores for these indicators, or indicators based on area of residence have also been used. Their use in Australian research and the specific advantages and problems of each have been examined by Najman (1988) and Turrell et al. (1994). In Britain, the examination of socioeconomic differentials in health status has been based

traditionally on occupation, while in the United States, educational and area based indicators have been more common.

In the review of inequalities in health which follows, the terms 'social class' and 'socioeconomic status' have been used interchangeably depending on the terminology of the author(s).

British evidence

The first systematic study to link social conditions and mortality is attributed to Villermé, a French physician who, in 1826, contrasted the death rates of various rich and poor areas in Paris. This work was subsequently expanded in British studies by Chadwick and Farr (Shryock 1979), and by the early 1900s the inverse association between social class and mortality was so well recognised in Britain that it contributed to the development of the Registrar General's Classification of Occupation (Social Class) for analysis by class stratification. Concern about social inequality and health remained during the 1930s and 1940s, especially the issues of children's health and geographical inequity in the provision of health services, and led to the establishment in 1948 of the British National Health Service (Blaxter 1991). Subsequent concern that differences between population groups might not, as anticipated, be decreasing and the realisation that Britain was failing to match the improvements in other developed countries, for example in life expectancy gains, led to the establishment of the Working Group on Inequalities in Health, chaired by Sir Douglas Black, and the publication of The Black Report in 1980 (Black 1980).

Although evidence of a strong link between social class and mortality was not new, the debate was rekindled[1] by the findings of the Report which concluded that, while genetic and cultural or behavioural explanations play their part, the predominant or governing explanation for inequalities in health lay in material deprivation. The Working Group's analysis of British mortality data for 1970-72 again showed that mortality tended to rise inversely with falling occupational status for most causes of death, including cardiovascular disease. The Working Group also found that the risk of death for men in each occupational class was almost twice that of women. There were regional differences also. The healthiest part of Britain appeared to be the southern belt below a line drawn across the country from The Wash to the Bristol Channel,[2] and the Report

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[1] Political and public health reactions to The Black Report, and to The Health Divide which followed, are described in detail in Townsend P, Davidson N, Whitehead M. (eds) *Inequalities in Health: The Black Report and The Health Divide* : London, Penguin Books, 1988, and also in Strong (1990).

[2] The premise of a north/south health gradient in Britain requires qualification. The regional divide in mortality is not static but changes over time and has narrowed for infancy, childhood,

suggested that social factors, including industrial and occupational, were implicated. The Report's most controversial claim was that health inequalities had widened during the preceding decades despite the fact that the National Health Service had been introduced to equalise health care access and outcomes.

The report also considered medical consultation and hospital admission rates. Inequalities were more pronounced for chronic than acute or short-term ill health. The class gradient for standardised patient consultation ratios for hypertension was steeper than that for mortality and the Working Group suggested that this indicated more severe sickness or a smaller likelihood of treatment with declining social class. The data are difficult to interpret, however, because the rates most likely reflect not only the incidence of the disease, but also the propensity of an individual to seek treatment and the subsequent medical response. The authors also drew attention to the fact that insufficient attention was being paid to the need to promote healthier lifestyles more equally among the population, and that there were financial, educational, and other restrictions on the opportunities of some groups to achieve better lifestyles which tended to sharpen health inequalities in Britain.

The report attracted criticism in relation to its methodology, assumptions and quality of data it used and these are discussed later. The debate surrounding the report proved to be the catalyst for a large number of studies directed towards clarification of the relationship between socioeconomic status and health outcomes and these have been reviewed by Whitehead (1987), Smith et al. (1990), Townsend (1990b), Morris (1990) and others. The studies overcame many of the criticisms made of the Black Report by using alternative datasets and methodologies, and substantiated the authors' original findings. For example, in order to minimise the effect of numerator-denominator bias in calculating mortality rates from two independent data sources, Marmot and McDowell (1986) considered only two broad occupational groupings. Using data from the 1979-83 Decennial Supplement on Occupational Mortality they demonstrated that for coronary heart disease and for stroke, non-manual workers had a lower risk of death than manual workers and that this social gradient had widened over the previous decade. Coronary heart disease mortality in non-manual workers had declined in every part of Britain and, by contrast, standardised mortality ratios in manual workers had stayed roughly constant in every region but Wales.

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adolescence and early adult years (Illsley and Le Grand 1993). Also, small area analysis shows there is large variation within regions that seems to be linked with social deprivation (Whitehead 1987; Britton 1990; Phillimore et al. 1994).

Evidence suggests that heterogeneity in the national data produces underestimates of the social class gradient. The Whitehall study (Marmot et al. 1984) followed the mortality of some 17,000 office-based civil servants over a 10-year period and showed that the mortality rate from coronary heart disease among the lowest grade was three times that of the highest grade. This was true also for a range of other causes. A study of mortality from coronary heart disease in the British Army in 1973-79 produced similar results with a sixfold difference between extreme ranks (Lynch and Oelman 1981). These estimates are larger than the mortality differences nationally between Registrar General's classes I and V and suggest that improved categorisation of socioeconomic position produces greater mortality differentials.

Results from longitudinal studies enable socioeconomic differences to be assessed free of numerator-denominator biases. The longitudinal study of the Office of Population Surveys and Censuses comprises a one per cent sample of people enumerated at the 1971 Census of England and Wales in which vital events in the sample and their families have been continuously recorded. Results have confirmed the relationship between socioeconomic status and mortality risk for a variety of indicators including housing tenure and car ownership (Goldblatt 1990). Mortality differentials were demonstrated for coronary heart disease and stroke in men and women. Enriching the indicators of socioeconomic status beyond occupation has been valuable for examining differential mortality in women (Smith et al. 1990). The British Regional Heart Study followed-up middle-aged men for 6 years and found substantial social differences in the risk of coronary heart disease with the manual social classes having higher prevalence of coronary heart disease at screening and, subsequently, higher attack rates. Cigarette smoking was found to be the single most important factor accounting for these differences (Pocock et al. 1987) but manual workers also had higher levels of blood pressure, obesity and leisure time inactivity. Adjustment for these risk factors did not eliminate the gap between manual and non-manual workers in heart attack rates.

It would appear that risk factors are an important but incomplete explanation of the socioeconomic gradient in cardiovascular disease. The Whitehall study (Marmot et al. 1984) showed that the standard coronary risk factors could not completely account for the coronary heart disease differences between the grades.[1] A random sample survey of morbidity and fitness (Blaxter 1987) conducted in 1985 revealed social class differences in health and lifestyle.

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[1] It is perhaps unrealistic to expect single risk factor measurements to predict 10-year coronary mortality. More precise measurement of risk factors (exposure) might have strengthened the relationship.

Smoking and obesity were more prevalent in manual social classes, as was poorer fitness. However, the relationship was not always simple. Whatever their social class, smokers were found to have a poorer lung function than non-smokers, but the deterioration in lung function associated with smoking was greater in manual social classes than in non-manual. In a subsequent more detailed analysis, this last observation was modified. The analysis confirmed that risk factor behaviour, particularly smoking, was relevant to health but that behaviour had most effect when the social environment was good: rather less if it was already unhealthy (Blaxter 1990, p233).

Socioeconomic differentials in Britain in biomedical and nutritional indicators are not as clear as those for behavioural risk factors. Between October 1986 and August 1987, the Office of Population Censuses and Surveys conducted a dietary and nutritional survey of British adults which collected data on nutrient intake, body mass, blood pressure and blood lipids. Women in the lower social classes had the higher body mass index but there was no consistent trend with social class in men. Blood pressure, serum total cholesterol and high density lipoprotein cholesterol concentrations were not significantly related to social class in either men or women, nor were there significant differences between those working and those unemployed for either sex (Gregory et al. 1990).

Australian evidence

Australia does not have the history of research into the association between socioeconomic inequalities and health as does Britain, and for many years has relied on overseas reports for its documentation. Socioeconomic differentials in mortality during the 1970s were demonstrated repeatedly in Australia (Dasvarma 1980; McMichael and Hartshorne 1980; McMichael and Hartshorne 1982; Taylor et al. 1983; Gibberd et al. 1984; Dobson et al. 1985; McMichael 1985; Siskind et al. 1987), and some analyses gave evidence that inequalities were widening (Taylor et al. 1983; Gibberd et al. 1984; Dobson et al. 1985). As in Britain, greater attention has been given in recent years to socioeconomic differentials in health indicators other than mortality, and to understanding their causes. Also as in Britain, Australian researchers have tended to use occupational classifications to indicate socioeconomic status.

One of the first demonstrations of socioeconomic gradients in mortality from coronary heart disease and stroke was contained in an analysis of mortality by area of residence in Victoria during 1969-73. Powles and Birrell (1977) ranked metropolitan regions of Melbourne by socioeconomic indicators (eg proportion of business and administrative occupations) and showed clear inverse gradients for men and women aged 15-64. In an analysis of national data in

1980, McMichael and Hartshorne considered cardiovascular and cancer mortality for 1968-76 in middle-aged men for fourteen selected occupational groups and showed a coherent pattern of death rates in relation to indicators of alcohol consumption and smoking. Mortality rates for coronary heart disease and stroke displayed a socioeconomic gradient with rates highest among labourers.

Gibberd and colleagues (1984) analysed mortality from coronary heart disease for the period 1969-1978 by place of residence, occupation and country of birth, and found large variations among subpopulations. They found wide regional differences with a declining gradient from eastern to western Australia. Temporal declines did not vary greatly between the regions suggesting that the factors responsible for the change occurred throughout Australia. In a subsequent analysis, Dobson et al. (1985) demonstrated that, over the same period, mortality declined by about 25% in men aged 25-64. The greater declines occurred in the professional occupations, while lower socioeconomic groups had higher mortality rates at the beginning of the period and experienced smaller declines. The authors included an analysis of data from the 1980 Risk Factor Prevalence Survey which revealed lower risk factor levels in the higher status occupation groups for blood pressure, triglycerides, cigarette smoking, body mass and exercise but not for cholesterol. They suggested that the reduction in coronary heart disease was associated with multiple risk factors; however, no account was taken of the lag between risk factors and mortality and the authors were not able to look at trends in risk factors.

In 1985, McMichael investigated the relationship between mortality of Australian males aged 15-64 and social class, using the Congalton 4-point occupational prestige scale, for the peri-censal periods 1970-72 and 1975-77 combined. He found that age-standardised death rates generally were higher in the lower social classes. There was some evidence of a class gradient in mortality from coronary heart disease. Death rates tended to increase moderately with decreasing social class. The mortality gradient for stroke was a little stronger than for coronary heart disease. Ethnic variation between social classes could have been a confounding factor.

Reliance on occupation on the death certificate as the indicator of socioeconomic status has meant that data for women has been scarce.[1] Socioeconomic gradients for women as well as men were demonstrated by Siskind et al. (1987) using an area based socioeconomic indicator for Brisbane

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[1] The problem of assigning a class (socioeconomic) position to women is discussed in Turrell et al. (1994).

city residents. The results were similar for men and women: mortality rates for coronary heart disease and stroke during 1976 to 1979 were higher in lower ranked socioeconomic strata, which suggested that occupation *per se* was not the major factor in class differences.

Mathers (1994b) recently reported marked differentials in a comprehensive set of health indicators covering mortality, disability, self-perceived health, risk factors, health service use and preventive screening services, using a variety of measures of socioeconomic status (education level, occupation, occupational prestige, equivalent family income and socioeconomic disadvantage of area of residence). Working age men and women (25-64 years) with lower socioeconomic status had higher death rates and reported higher levels of illness and consequent reduced activity. The poorer health status of the socioeconomically disadvantaged largely explained their greater use of primary and secondary health services at the broad national level. Those of low socioeconomic status made less use of preventive and screening services and were more likely to have lifestyle risk factors. A subsequent analysis showed similar inequalities among older Australians (aged 65 or more), albeit less marked than for working-age people (Mathers 1994a).

The National Health Strategy initiative, a two year review and strategy development program established in 1990, had as a major objective

> 'to improve the equitable impact of the health system. As poor health is linked to socioeconomic circumstances, the Strategy will work to reduce inequalities in terms of cost, access and health status' (Macklin 1990).

In 1992, after the publication of a series of background and issue papers, a substantial research report was published on how income and environment affect health (National Health Strategy 1992a,b). Whitehead et al. (1993) likened the Australian report to Britain's Black Report, but contrasted its political reception. Neither the research findings of the 1980s nor the release of this report generated the political debate witnessed in Britain, which has continued to the present time. The report concluded that, in order to reduce health status inequalities by improving people's social and economic circumstances, policy needs to focus on five broad areas: the distribution of economic resources; education; living conditions; access to and conditions of work; and the provision of social support.

Evidence from the United States

A partial review of literature since the 1970s[1] describing the association between socioeconomic status and health for the United States is given by Feinstein (1993), who begins by describing the 'pioneering epidemiologic work' of Kitagawa and Hauser published in 1973. Their analysis of the 1960 Matched Records Study demonstrated a persistent inverse monotonic relationship between socioeconomic status and mortality from heart disease among men and women, which was generally stronger among persons aged 25-64 than among those aged 65 or more. This inverse association persisted whether educational attainment or household income was used as the indicator of socioeconomic status. Multivariate modelling demonstrated that the effects of education and income were largely independent. Their analysis of the Chicago Area Study[2] showed a sharp negative relationship between class and mortality between 1930 and 1960 for men and women which was relatively stable over time (in contrast to the British experience). The Chicago study used aggregated small area data and is subject to the limitations of ecological analysis. The results may also be confounded by the effect of reverse causality whereby sicker individuals move to poorer areas. While neither analysis took other variables such as risk factors into account, their analyses are seen as compelling evidence for the existence of differential mortality by socioeconomic status over this period (Feinstein 1993).

The analysis of trends in socioeconomic inequalities in mortality was extended from 1960 to 1970 by Yeracaris and Kim (1978). Their analytical approach was similar to that of the Chicago data by Kitagawa and Hauser (1973) except that the socioeconomic status of each census tract was based on a weighted index of four indicators for three selected metropolitan areas. The results suggested increasing socioeconomic differentials over time in mortality from heart disease and the authors made two interesting observations: when mortality is declining, socioeconomic differentials for heart diseases tend to increase; and, improvements in the nation's health tend to benefit higher socioeconomic groups and health deterioration to tax lower socioeconomic groups. They noted that male mortality was more susceptible to these observations than female mortality.

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[1] A review of earlier literature describing the association between social class and cardiovascular disease is given in Antonovsky A. Social class and the major cardiovascular diseases. *J Chronic Dis* 1968;21:65-106.

[2] A study of census tracts in the Chicago metropolitan area and surrounding suburbs for 1930, 1940, 1950 and 1960. Census tracts were ranked by socioeconomic status based on median rental payment, and linked with age standardised mortality rates.

In 1980 Kraus et al. demonstrated an inverse link between socioeconomic status and *risk* of coronary heart disease. Their analysis was based on risk factor data from male volunteers in a screening program for the Multiple Risk Factor Intervention Trial.[1] Data for blood pressure, cholesterol and cigarette smoking were combined into a composite risk score based on a multiple logistic equation from the Framingham Heart Disease Study. The finding of an inverse association was consistent with that from the Oslo study (Holme et al. 1976), which was possibly the only other study at the time to have evaluated risk of a coronary event as a composite score. The authors suggested that the popular notion that the risk of coronary heart disease was a problem largely among the affluent would have to be modified.

The analysis by Yeracaris and Kim (1978) suggested that the national decline in coronary mortality, which began about 1968 in the United States, did not occur simultaneously in all sectors of the community. During the 1980s, Wing and colleagues used geographic variation in the timing of the decline to examine socioeconomic characteristics associated with the onset of the decline. Using 507 State Economic Areas as the unit of analysis, they demonstrated considerable geographic variation in the timing, with metropolitan areas generally experiencing the decline earlier than non-metropolitan areas (Wing et al. 1986). Then an association was demonstrated between community occupational structure and coronary heart disease in 3102 counties using the proportion of workers in white-collar jobs as the indicator (Wing et al. 1987). This analysis suggested that the inverse socioeconomic gradient emerged in the United States when mortality began its national decline, and that the decline was initially more rapid in areas within higher levels of occupational structure, a finding consistent with late onset in many non-metropolitan areas (Wing et al. 1986). It was subsequently shown that areas with the poorest socioeconomic conditions were more likely to have experienced later onset (post 1968) than areas with the highest levels, and that income-related characteristics could account for most of these differences in timing between metropolitan and non-metropolitan areas (Wing et al. 1988). The authors saw the importance of community socio-environmental characteristics not as competing with risk factor explanations, but as providing the context in which positive changes in risk factors and medical care were possible.

The association of poverty *per se* with poor health was examined by Haan et al. (1987) who found that residence in areas characterised by a broad range of

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[1] A randomised primary prevention trial to test the effect of a multiple factor intervention program on mortality from coronary heart disease in 12,866 high-risk men aged 35-57 (Multiple Risk Factor Intervention Trial Research Group 1982).

social and environmental deprivations was prospectively associated with increased risk for all-cause mortality, even after adjustment for potential confounders. Their analysis compared the nine-year mortality experience (1965-1974) of Oakland residents who lived in federally designated 'poverty' areas with those who lived elsewhere in Oakland, California. The increased risk of death of those who lived in poverty areas persisted after adjustment for age, sex, race, baseline physical health status, low income, lack of medical care, unemployment, education, health practices, social isolation, or psychological uncertainty or depression. If it is assumed that poor measurement of these variables did not affect their power to discriminate, the results support the hypothesis that properties of the social and physical environment may be important contributors to the association between low socioeconomic status and excess mortality, and that the association is independent of some factors thought to contribute to the link between socioeconomic position and disease.

Evidence of trends in socioeconomic differentials in mortality were documented by Feldman et al (1989) by comparing estimates from the 1960 Matched Records Study with 1971-84 data from the first National Health and Nutrition Examination Survey Epidemiologic Followup Study. When educational level was used as the indicator, differentials increased among men due to more rapid declines among the more educated than the less educated. Among women death rates declined at about the same rate regardless of educational attainment so that the strong inverse relationship between education and mortality was maintained. Trends in educational differentials for heart disease contributed significantly to these patterns in total mortality.

Jarjoura and Logue (1990) used the percentage of employed persons in lower socioeconomic strata to investigate the relationship between socioeconomic status and heart disease mortality in 1200 census tracts in the counties of Ohio. The results showed mortality rates increased monotonically with decreasing socioeconomic status. The paper's primary significance is in providing more appropriate methodology for the estimation of rate ratios and their confidence intervals.

In 1993, Pappas et al. duplicated the analysis of Kitagawa and Hauser (1973) of differential mortality in 1960 using records from the 1986 National Mortality Followback Survey and the 1986 National Health Interview Survey. The inverse relationship between socioeconomic status persisted in 1986 and was stronger than in 1960; that is, disparity in mortality rates according to income and education increased in the United States between 1960 and 1986 for men and women.

Some evidence from other countries

Leclerc (1989) compared differential mortality by cause of death for selected European countries by comparing mortality among male unskilled workers with that of 'all occupied men' using mortality estimates from cohort studies based on a census and a 10 year follow-up (5 years for France). Inequalities in mortality from cardiovascular disease were demonstrated for Finland, Norway, Denmark, England and Wales, and France, with the largest differential observed in Finland. Lynge et al. (1989) compared occupational groups in Norway, Sweden, Finland and Denmark and derived mortality estimates from linkage studies between the 1970 census and 10-year death records. Cardiovascular deaths among men engaged in 'pedagogical' work were lower than 'expected' in each Nordic country.

An inverse relationship between cardiovascular disease and level of education has been found for Denmark, Finland, England and Wales, Hungary, Norway and Sweden (Valkonen 1989b). The relationship was mainly due to the difference between those with less than 12 years and those with at least 12 years of education. In all countries, educational differences in cardiovascular mortality were greater among women than men. Only among men in England and Wales was the change in the inequality coefficient between 1971-75 and 1976-80 for cardiovascular disease statistically significant. The widening inequality was due to an increase in mortality in the lowest educational category and a decline of 20-40% in other educational categories.

Summary

Socioeconomic inequalities in cardiovascular mortality have been documented for the United States, Britain and other European countries using a variety of indicators. In the United States and Britain inequalities have probably increased over recent decades. The evidence suggests that inequalities also widened in Australia during the 1970s but there are no later data. Inequalities have been demonstrated for a variety of socioeconomic indicators and it is evident that socioeconomic status is substantially implicated in cardiovascular health, although the formal criteria for causality are not fully met (Kaplan and Keil 1993). None of the socioeconomic indicators is likely to cause health outcomes directly but rather they are markers for a complex set of social conditions and processes which may be causally implicated (Macintyre 1986). The following section examines the conditions and processes that might be intervening between socioeconomic measures and health outcomes.

2.7 Potential explanations for socioeconomic inequalities

The consistency of the evidence within developed countries for differentials in mortality and morbidity has been the catalyst for much research and debate concerning possible explanations for the patterns observed and there are numerous reviews of the literature (Kaplan and Keil 1993; Blaxter 1991; Strong 1990; Townsend 1990a; Macintyre 1986; Hart 1986; Pamuk 1985). Explanations for the association between health and socioeconomic status are often addressed under the classification used in the Black Report: artefact (statistical or methodological); social selection; cultural and behavioural; and materialist and structural. The discussion which follows is not solely directed to cardiovascular disease and relies heavily on British research, nevertheless the principles and arguments espoused are relevant to explaining socioeconomic inequalities in cardiovascular disease in Australia.

Statistical or methodological artefact

Advocates of the 'artefactual' explanation criticise the methods used by the authors of the Black Report on several grounds (Illsley 1986; Carr-Hill 1990; Strong 1990), especially their reliance on the British Registrar General's Classification of Occupations as an indicator of social class (Jones and Cameron 1984). Much of the research literature has relied on occupation as an indicator of socioeconomic status, partly because of the availability of data. However, differentials of the same magnitude have also been demonstrated for other indicators such as education and income and the common finding has been that the less affluent and educated of society are the least healthy. By restricting the analysis to occupations which could be consistently identified throughout the period 1921 to 1972, Pamuk (1985) demonstrated that revisions to the Registrar General's classification did not appear to be the source of widening in mortality differentials. There are problems of numerator and denominator biases associated with estimating death rates for occupational groups using data from death registrations and population censuses. However, the net effect of these has been demonstrated to be minimal by studies which have avoided such issues but found the same general pattern of differentials (Fox et al. 1990; Marmot and McDowall 1986).

Indicators of inequality which use the ratio of mortality in extreme social classes have been criticised for omitting significant proportions of the population and not taking the relative sizes of the classes into account. Using composite measures of inequality,[1] it has been demonstrated that the rise in

[1]Koskinen (1985) used a modified Index of Dissimilarity to calculate the minimum number of deaths that would have to be redistributed in order to give the same age standardised death rate in each class. Pamuk (1985) used the Slope Index of Inequality which can be interpreted as

inequality in adult mortality in Britain was not an artefact of changes in the Registrar General's classification nor of changes in the population distribution between occupational classes (Koskinen 1985; Pamuk 1985). It is also relevant that differentials have been demonstrated for indicators of health other than mortality. Differentials are wider if measured in terms of potential years of life lost because deaths among lower socioeconomic groups occur at younger ages (Blane et al. 1990). Concern that health and illness differentials based on self-reported data result from variation in perceived norms between socioeconomic groups has been partially addressed by the consistency shown between some self-reported data on health and symptoms, and more objective physiological indicators of morbidity such as lung function and blood pressure (Blaxter 1990).

There has been little systematic research conducted on the effect of variations in diagnostic fashion over time on apparent variation in disease or mortality, or the possibility that patients may be diagnosed or treated differently according to their social position (Macintyre 1986). It is possible that diagnosis of cause of death may have been influenced by the diagnostician's knowledge of the social class or other demographical characteristics of the person involved and that this may have contributed to the pattern of socioeconomic differentials over time. However, the quality of official mortality data for coronary heart disease (ICD9 410-414) has been validated in Australia in recent years and found to be reasonably accurate and reliable (Dobson et al. 1983; Sexton et al. 1992).

In summary, the analysis of health related statistics over time has to contend with variation in data collection practices, definitions, coding practices, imperfections in measuring instruments and other sources of bias. Nevertheless, the body of evidence to date suggests that trends in socioeconomic differentials in mortality and morbidity are not an artefact of statistical methodology. Indeed, the heterogeneity that exists within most occupational classifications is likely to produce underestimates of the association between socioeconomic status and health however measured.

Social selection

It has been suggested that continuing or widening socioeconomic inequalities in health, whether measured by occupation, income, educational attainment or other indicators, may be the result of the healthy moving up the socioeconomic scale and the unhealthy moving down. This explanation assumes that class differences are produced by individual inequalities of personal health, fitness,

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the average decline in the age standardised death rate as one moves from the lowest class to the highest.

intelligence and ability and that social and economic privilege is earned by people with greater personal physical and intellectual resources (Hart 1986). It asserts that social mobility reinforces health inequality by widening the gap in survival chances between rich and poor, and social class becomes a distributional measure of inherited ability. This is an important issue because it suggests that, to some extent, inequalities in health are not attributable to structural socioeconomic inequalities (Wilkinson 1986). It suggests that people are in lower classes because they are less healthy rather than people are less healthy because they are in lower classes. Concern over this issue led Stern (1983) to conclude that the measurement of change over time in class gradients in health should be of class origin rather than of achieved social class.

The selection hypothesis received support from the research of Illsley on the height of women, reproductive outcomes and interclass mobility at marriage (Illsley 1955). Taller women were demonstrated to be upwardly mobile and produced babies with a higher rate of survival (Illsley 1986). There is also evidence that health in early life is associated with socioeconomic differences in adulthood, either by maintaining early inequalities or by selective mobility (Wadsworth 1986; Power et al. 1990; Fogelman et al. 1989). However, while the evidence showed that health and social mobility were associated during adolescence and young adulthood, social inequalities in health were equally evident among the non-mobile (Power et al. 1990; Fogelman et al. 1989).

Data from 10 years' follow-up of mortality in the OPCS Longitudinal Study suggest that health related selective mobility is not an important determinant of socioeconomic differentials (Fox et al. 1990). It may even have the opposite effect by removing unhealthy people in the lower socioeconomic groups from the workforce, and hence outside the classification of occupations, before they die (Blaxter 1991). The net effect of movement between social classes between the 1971 and 1981 censuses on class gradients in male mortality was found to be negligible. It is argued that the existence of sharp gradients at ages over 75 years, similar to those at younger ages, suggest that selective health related mobility between social classes does not contribute to differentials in mortality for men over 50 years (Fox et al. 1990). Reanalysis of the Whitehall Study data confirmed that selective social mobility did not explain the mortality differentials in that study (Davey Smith and Shipley 1990). The mortality gradient was maintained even ten years after the original classification by employment grade. Excluding those with identifiable disease at baseline did not greatly affect mortality differentials. Studies of the rate of social mobility in Britain have shown only very modest increases, and that even if a substantial proportion of this increase was related to the process of health selection, this

could have had only a marginal effect on the growing gap in mortality differentials (Wilkinson 1986). Hart (1986) argues that the chronological pattern of class gradients (namely widest in the age group before social mobility has peaked, a narrowing in middle age and persistence into retirement) is inconsistent with a major role for social selection in the creation of socioeconomic inequalities. The fact that differentials occur for educational attainment, which does not change with decreasing health status, also suggests that the effect of health-related mobility is not great, certainly not past young adulthood.

In summary, social selection appears not to be a major cause of health differentials between socioeconomic groups (Blane et al. 1993) but its importance may be more relevant at some life stages and for certain population groups. In relation to the size of the differentials observed, its contribution is probably always small (Wilkinson 1986). An independent multidisciplinary committee supported the view that factors related to social mobility 'make only minor contributions to the observed class differences in health, and there is no evidence that their influence has increased.' (Smith and Jacobson 1988).

Behavioural and cultural explanations

Behavioural explanations portray the lower health status of lower socioeconomic groups in terms of the adverse lifestyles of its members. For example, Najman (1994) has argued that since class (or socioeconomic status) exists only as an abstraction, it cannot be the direct or proximate cause of disease or death. With health care relatively freely available to all,[1] lifestyle differences between socioeconomic groups may have become the major basis of class differences in health and illness. Those of lower socioeconomic status have both a higher prevalence of disorders which are known to be linked to specific risk factors, and a higher prevalence of those risk factors including behaviours such as cigarette smoking, excessive alcohol consumption, adverse food habits and inactivity. Further, these lifestyle differences are (arguably) a reflection of the education and income inequalities in society, and occupational class only appears to be important because it is a function of a person's education and income. It is education and income which are more directly associated with knowledge and with opportunities to make lifestyle choices which are of direct relevance to health. Najman (1994) concludes that one of the major reasons why persons who are of lower class have higher rates of disease and death is that they lead less healthy lifestyles.

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[1] Government policy implemented in Australia since 1970.

Various reasons have been speculated as to why lower social class and less healthy behaviour are associated. One 'individualistic' view is that this lifestyle might reflect a value system which ranks instant gratification higher than deferred gratification, unlike the middle classes. This approach may also assume that detrimental behaviours result from a lack of knowledge of personal heath care and that beneficial behaviour modification will follow the provision of preventative health education. Such 'individualistic' views give little weight to structural, cultural and societal influences in shaping personal beliefs and actions and fail to recognise that collective group ideologies and practices can outweigh personal costs and benefits (Hart 1986).

Hart (1986) uses cigarette smoking to illustrate the influence on behaviour of factors which are 'external' to the individual, and suggests several reasons as to why people of lower socioeconomic status in Britain have been so reluctant to stop smoking despite health education, curbs on advertising, health warnings on packets and increasing tax.[1] If increasing consumption among women and a reluctance of manual workers to cease smoking is related to stress release from the tension of managing work and home or the physical stress of manual work, then the issue is one of material deprivation rather than flawed behaviour or cultural backwardness. A cultural view would note that smoking has been a socially valued behaviour which was encouraged in two world wars and it has long been a symbol of greater personal independence among young people. Smoking remains closely interwoven with the culture of the manual working class and a cultural redefinition of the behaviour is required in this group if health education is to be effective. Hart speculates that manual workers may have a greater need of external symbols of status change because they face the transition from school to work at a younger age.

Similar considerations apply to other behaviour such as physical exercise, alcohol consumption and diet. For example, diet is profoundly influenced by cultural or local social customs, informal and formal education, the availability and price of goods in local markets, advertising, recipes and fashions recommended by the media, and decisions taken by farmers and the manufacturers of food products as well as by government (Townsend 1990a). Clearly, lifestyle behaviours are far from being activities determined solely by individual choice. The ability to respond to health education depends upon the strength of practical and social barriers and on the differential capacity of people to exert control over their lives (Blaxter 1991).

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[1] The same preventive actions occurred in Australia during the 1980s.

Health education aimed at the personal modification of lifestyles might be resisted if the power to change was, or was felt to be, unavailable (Blaxter 1990, p243). The middle classes are known to be more mobile and hence may be more willing to embrace new ideas because their lifestyle requires adaptation to new localities, jobs and relationships (Hart 1986). In this context group solidarity and peer pressure become less important. Behaviour change implies opportunity, self assurance, personal autonomy and control over circumstances which flow from the opportunity for education, occupational choice, personal growth and development. Such opportunity, it is argued, is determined by the distribution of wealth and income (Hart 1986). Inequalities of material resources directly reinforce forms of deprivation which are expressed in ideas and behaviour which appear as cultural norms. Individualistic explanations of health related behaviour may be more appropriate for understanding middle class responses to health education than explaining why lower socioeconomic groups have proved more resistant to change. Since higher socioeconomic groups have been most likely to respond to health education messages to improve lifestyle, broadly based population strategies which focus on individual patterns of behaviour can have the effect of widening socioeconomic differentials in health (Blaxter 1991). Also, the adoption of a healthier lifestyle by people in higher socioeconomic strata may have the inadvertent consequence of increasing the pressure placed on other social groups to increase their consumption of unhealthy products (Beaglehole 1990). An example is young women who became the focus of attention of the tobacco and alcohol industries.

While lifestyle differences and related risk factors are undoubtedly important, they may not be the major reason for socioeconomic differentials in health in Britain. The Whitehall study of civil servants found that the inverse association between employment grade and coronary heart disease mortality was only partially explained by adjustment for the standard coronary risk factors (Marmot et al. 1984). It is likely that more precise measurement of risk factor exposure would have increased their explanatory power; nevertheless social gradients were also observed in diseases unrelated to those risk factors, and this suggests that other factors were operating (Marmot 1989). Very detailed analysis of the British Health and Lifestyle Survey[1] demonstrated the well known inverse relationship between major health-related behaviours and socioeconomic indicators but also found that the relationship was not straightforward. 'If circumstances are good, "healthy" behaviour appears to

[1] A national cross-sectional sample survey of men and women aged 18 or more living in private households in England, Wales and Scotland. Data collection occurred during 1984-85 and over 9,000 responded, a response rate of 73.5%.

have a strong influence upon health. If they are bad, then behaviours make rather little difference' (Blaxter 1990, p216). While acknowledging the difficulties of distinguishing the health effects of socioeconomic status from lifestyle because of the influence each exerts on the other, it was concluded that life circumstances (socioeconomic, external and psycho-social) were more important determinants of 'everyday' health than healthy or unhealthy behaviours (Blaxter 1990). The cross-sectional nature of the study meant that nothing could be inferred about the role of lifestyle in the development of future disease except to the extent that it is foreshadowed in the experience of poorer current health.

Various small scale (often qualitative) studies suggest that the social setting enables or constrains the practice of health-related behaviours by psychological mechanisms such as lay conceptions of health, health locus of control, and attitudes towards specific health-related behaviours (Calnan 1989; Calnan and Williams 1991, Mullen 1992; Owen and Bauman 1992). The 'psychological' literature also provides various models to explain health behaviour as a rational action (Carter 1991). Although the notion of health behaviour is very individualistic, even an 'individualistic' model such as the 'Theory of Reasoned Action' (Ajzen and Fishbein 1980) has a social component. This model sees the strength of a person's intention to perform a specific behaviour as a function of two factors: attitude towards the behaviour and the influence of the social environment or general subjective norms on the behaviour. However, in empirical tests of the model, the focus has been on the links between attitude and behaviour and the social component of the model has been rarely addressed.

Materialist and structural explanations

Materialist and structural explanations for socioeconomic inequalities in health focus on the external environment. Included are factors such as access to economic resources, conditions under which people live and work, social support and access to health care.

Difference in access to economic resources, usually measured by income, can contribute to health differentials through inability to purchase goods and services that influence health. Low income was strongly associated with poorer health in the Health and Lifestyles survey (Blaxter 1990). Wilkinson presented data from the survey which suggests that the relationship between income and health is non-linear; and that health[1] improves as income increases from the lowest to middle incomes with no further gains in health expectancy for higher

[1] As measured by disease and disability, illness and psychosocial health.

incomes (Wilkinson 1992). In Australia, family income has been associated with large differentials in a range of illnesses, chronic conditions and risk behaviours (Mathers 1994b).

Differences in living conditions such as poor sanitation, overcrowding, poorly ventilated or damp housing, proximity to hazardous industrial or commercial waste, air pollution, unsafe food or water supplies may also contribute to health differentials. It has been claimed that public health measures and the general rise in the standard of living have contributed more to the improvement of population health than specific medical interventions, either curative or preventive (McKeown 1976). Nevertheless, real differences remain. In Australia, poor physical environments are believed to be an important contributor to the poor health of Aboriginal people. Aboriginal death rates from cardiovascular disease are 2.5 times those for the Australian nation. A number of deaths of Aborigines living in more remote areas has been attributed to chronic rheumatic heart disease, which is an uncommon cause of death for other Australians (Australian Institute of Health and Welfare 1992). The disease is thought to be linked to adverse living conditions, associated with poverty, which are conducive to the spread of streptococcal throat infections, such as poor housing and hygiene (Brundtland 1994; Anonymous 1982).

Poverty and adverse living conditions in the early stages of life may be linked with coronary heart disease in middle age; however the evidence is in dispute. Barker and colleagues advocate the view that coronary risk is strongly influenced by deprivation in fetal and early infant life and that biological structures are programmed at this time which endure into adulthood (Barker 1992; Barker and Osmond 1992; Fall et al 1995a; Fall et al 1995b). In this hypothesis, undernutrition during pregnancy is believed to be an important factor (Barker et al. 1993; Barker 1995). The epidemiological evidence concerning early life experience and adult cardiovascular disease has been reviewed by Elford and colleagues (Elford et al. 1992; Elford et al. 1991). While all ecological studies described strong, dose related relationships between adult cardiovascular mortality and an indicator of early life experience, they failed to satisfy several established criteria for causality. In longitudinal and case-control studies, no consistent dose-response relationship was found between the indicator of early life experience and adult cardiovascular disease (Elford et al. 1991) and relationships were usually not specific to cardiovascular disease. Inadequate treatment of confounding by socioeconomic disadvantage was a common problem in all studies. The reviewers concluded that these studies do not provide strong support for the hypothesis that experiences in early life determine the subsequent risk of cardiovascular disease. Recent studies of data

from Finland (Lynch et al. 1994) and from England and Wales (Ben-Shlomo and Davey Smith 1991) which have allowed for the confounding effect of socioeconomic status over time have produced results which suggest that socioeconomic conditions in adulthood are more important predictors of coronary mortality than socioeconomic conditions in childhood. Baker et al. (1993) found no evidence of a birth cohort effect in the decline in coronary mortality, a finding which is more compatible with theories involving contemporary lifestyle changes than conditions in early life. The complexity of the issues and methodological problems make this a difficult area for epidemiological research (Elford et al. 1991; Baker 1994). Suggestions for future research involve refining the measures of exposure, adjusting for correlates, and more specific formulation of the hypothesis (Paneth and Susser 1995; Paneth 1994).

The working environment can affect health directly through factors such as exposure to physical and chemical hazards or occupational injuries, or more indirectly through physiological factors related to manual work or shift work, or psychological and social factors including its stressful and alienating effects. Factors more typical of manual workers, such as low decision latitude at work, psychosocial strain, noise, shiftwork, monotony, time pressure, and few possibilities for growth, have been associated with increased risk of coronary heart disease (Hammar et al. 1994; Akerstedt et al. 1984; Alfredsson et al. 1982; Karasek et al. 1981). Hasan (1989) has suggested that the study of the development and satisfaction of human needs might be a more helpful concept in this context than 'stress', and that unsatisfied needs may lead to physiological states, activities and behaviours which increase the probability of disease and death. Pieper et al. (1989) demonstrated that some coronary risk factors may be related to psychosocial aspects of work, in particular the decision latitude of the job. Marmot and Theorell (1988) reviewed the contribution of work to the relationship between social class and cardiovascular disease. They concluded that part of the association may be due to psychosocial work conditions and that psychosocial work conditions may affect the risk through either neuroendocrine mechanisms[1] or lifestyle.[2] One pathway for the cardiovascular effects of stress is through hypertension. Although the evidence that psychosocial stress leads to raised blood pressure is unclear (Freeman 1990), a potential explanation of recent data is that genetic

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[1] Experimental research into the link between the brain and the immune system, and between emotional states (involving stress and lack of control) and disease states, is reviewed in Mestel R (1994) Let mind talk. *New Scientist* (23 Jul): 26-31. Links between neuroendocrine responses and cardiovascular pathology are discussed by McQueen and Siegrist (1982).

[2] For example, tobacco smoking may be undertaken to stay awake in jobs which involve boredom and lack of skill discretion, or to relieve stress in 'caring' occupations.

predisposition to hypertension is moderated by psychosocial experience (Anonymous 1994).

Social support is also included among the psychosocial factors which may contribute to differentials in heart disease. Lack of support may be considered as a social stressor contributing to the development of disease, or as a buffering mechanism in the association between a social stressor and strain experience. Health protective effects operate at the level of society (strong integration via cultural patterns), at the level of interaction behaviour between individuals (cognitive, emotional, instrumental assistance and positive feed-back) and at the emotional level (the feeling of security, lowered anxiety and aggression, better chances of relaxation) (Siegrist et al. 1986). Physiologically, social support should lead to a smaller increase of endocrine secretion of stress hormones (Siegrist et al. 1986). Studies which have demonstrated an explanatory role for social support have been reviewed by Siegrist (1991). The Alameda County study, for example, demonstrated that social networks and social support influenced health, and were differentially distributed by socioeconomic status ie. higher socioeconomic status had more social contacts (Berkman and Syme 1979). The association between social ties and all-cause mortality was independent of socioeconomic status, lifestyle behaviours and baseline health status. Social and community ties included marriage, contact with extended family and close friends, church membership, and other group affiliation.

The fact that socioeconomic inequalities have been demonstrated for a variety of diseases has led some researchers to speculate that a general explanation may be more appropriate than multiple disease specific explanations. It is argued that socioeconomic inequalities in health have a general explanation, and that the reason that those in lower socioeconomic groups consistently have higher rates of disease is, in part, due to compromised disease defences and increased general susceptibility (Syme and Berkman 1976). The emphasis is not so much on specific risk factors for specific diseases but more on general aspects of the living environment that compromise bodily defences and thereby affect health and well-being in general. The result is general susceptibility to a variety of diseases.[1] The general susceptibility approach is compatible with the risk factor approach. Syme and Berkman argue, for example, that one such factor might be stress and the ability to cope with life events. Coping styles are likely to be the product of environmental situations and can include the

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[1] Antonovsky (1989), arguing from the perspective of protective (rather than risk) factors, advocates the use of concepts such as 'generalised resistance resources' and 'sense of coherence' to help explain inequalities in health; concepts which seem to be complementary to that of 'general susceptibility'.

adoption of risk factor behaviour such as cigarette smoking. The general explanation and risk factor explanation which focuses on a set of specific causal factors, both seem to be relevant to the explanation of socioeconomic inequalities in cardiovascular disease (Marmot et al. 1984; Marmot 1986).

Lack of access to the health care system may contribute to the relationship between low socioeconomic status and poor health; however, its influence is generally regarded as less influential than other factors. Australian data (National Health Strategy 1992a,b) show that lower socioeconomic groups are more likely to use medical and hospital services than higher socioeconomic groups, although the differential is reduced when their poorer health status is taken into account. They are more likely to delay seeking treatment than higher socioeconomic groups and are less likely to use preventive services, early intervention and screening services, rehabilitation and after-care services. The cost of services can be a greater deterrent to the use of services by lower socioeconomic groups (especially for dental care). When analysing *health care* inequalities, Macintyre (1989) advocates a distinction between those inequalities that might generate inequalities in *health* in the first place (primary prevention), those that might influence cure or survival rates (secondary prevention), and those relating to rehabilitation or nursing care (tertiary prevention). A distinction is also noted between inequalities along a continuum (for example, income) and focussing on particular disadvantaged sub-groups of the population. In Australia these include, for example, Aboriginal and Torres Strait Islander peoples, the long-term unemployed, rural and remote communities, the elderly, ethnic groups and people of non-English speaking background. While data on differential use of heath services may be available, it is not possible to say what effect such differentials might have on differentials in health. 'Rarely, however, is the magnitude of the disparities in health care sufficient to explain the disparities in heath or death rates' (Macintyre 1989).

Summary

Mortality inequalities are unlikely to be the product of statistical artefacts. Factors relating to social selection probably make a small contribution to inequalities in adult life, although some believe their contribution is underestimated (West 1991). Risk factor differentials are undoubtably important contributors to cardiovascular mortality differentials as it also seems are socioeconomic factors, both indirectly through risk factors and directly through pathways as yet not fully understood. The role of socioeconomic circumstances and associated biological factors in fetal and early life on

cardiovascular inequalities in later life is unresolved. Psychosocial factors may also have a contributory role.

Materialist and structural explanations need not be seen as competing with cultural and behavioural explanations but rather as complementary components in the causal chain. All are closely interwoven and it has proven difficult to disentangle their relative effects on health outcomes. There is no doubt that the material conditions of life, and the social structure, influence behaviour, which in turn affects health. In addition, the influences on behaviour which are loosely grouped under the heading of culture, appear to affect patterns of eating, drinking, and social relationships that may be independent of current material conditions (Marmot et al. 1987). Clearly, for a fuller understanding of factors leading to increased risk of cardiovascular disease, it is necessary to consider lifestyle and biomedical risk factors in their socioeconomic and cultural context. To impose a dichotomy is likely to be misleading (Powles and Salzberg 1989). Medical care is not believed to be a significant influence on health inequalities among socioeconomic groups, either in their generation or reduction. However, studies which link usage with need and outcome have not been conducted.

It is clear that changes in lifestyle and lifestyle related risk factors are a function of socioeconomic level. A greater propensity among the higher socioeconomic groups to respond positively to health messages has the potential to contribute to widening inequalities in cardiovascular mortality. For example, in the United States, the more educated strata have made greater changes in eating, smoking and exercise habits than the less educated and this may have contributed to widening inequalities in cardiovascular mortality in that country. Chapters 7 and 8 of this thesis answer such issues for Australia. For example, 'Have the socioeconomic inequalities in cardiovascular mortality of the 1970s continued into the 1990s?', 'Has the gap widened?', and 'Which risk factors are most likely to have contributed to inequalities and trends in cardiovascular mortality?' are some of the questions addressed in Chapter 7. The issue of trends in inequalities in risk factors has rarely been considered and chapter 8 represents the first comprehensive analysis. It examines, for men and women, and for age groups, how Australians of different socioeconomic status changed their lifestyle risk factors over 1980s and whether they experienced different trends in biomedical risk factors.

2.7 In summary: thesis, hypotheses and aims

The literature review provided a context for the thesis and introduced some of its main hypotheses and aims. These may be expressed more fully as follows:

Thesis **that the cardiovascular risk factor profile of Australians improved during the 1980s, consistent with falling death rates, but important social inequalities remain.**

Hypothesis 1 **that the risk factor profile of Australians improved during the 1980s in line with the decreasing mortality rates for cardiovascular diseases.**

Aim 1 to examine changes in risk factor levels over time, for men and women separately, by age group.

Aim 2 to identify the particular risk factor changes which might help explain the continuing fall in cardiovascular mortality rates.

Aim 3 to identify any risk factors with an unfavourable trend.

Hypothesis 2 **that variation in blood pressure measurement technique between centres in repeat multi-centre studies[1] has minimal effect on cross-sectional and trend analyses**

Aim 1 to document the experience of the Risk Factor Prevalence Study in terms of departure from the measurement protocol and variation in measurement technique.

Aim 2 to estimate the effect of blood pressure measurement error on cross-sectional and trend analyses.

Aim 3 to assess the extent to which trends in blood pressure identified in Australia may have been due to variation in blood pressure measurement technique.

Hypothesis 3 **that immigrant groups in Australia which have a lower mortality rate from cardiovascular disease also have a beneficial risk factor profile.**

Aim 1 to compare the levels of biomedical and behavioural risk factors among a range of immigrant groups in Australia, using native-born Australians as a reference group, with special attention to those immigrant groups which have lower cardiovascular mortality, particularly immigrants from Italy and Greece.

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[1] Using normal mercury sphygmomanometers and following the MONICA protocol.

Aim 2 to assess the effect of duration in Australia on risk factor levels among immigrants.

Hypothesis 4 **a) that the socioeconomic inequalities in cardiovascular mortality of the 1970s continued into the 1990s;**

b) that inequalities in mortality are consistent with those in risk factors;

c) that socioeconomic groups which improved their risk factor profiles the most also experienced greater improvements in mortality;

d) that occupational inequalities in risk factors are not fully explained by differences in educational attainment.

Aim 1 to examine trends in socioeconomic inequalities in cardiovascular mortality, with reference to socioeconomic patterns in major cardiovascular risk factors, using occupation to indicate socioeconomic status.

Hypothesis 5 **that socioeconomic groups responded differently to the health promotion activities of the 1980s and experienced different trends in biomedical risk factors.**

Aim 1 to examine trends in socioeconomic inequalities in behavioural and biomedical cardiovascular risk factors using educational attainment to indicate socioeconomic status.

Hypotheses 1 to 5 are addressed in chapters 4 to 8 respectively.

Chapter Three

Survey methods, data issues and definitions

3.1 Survey methods

The primary source of data for this thesis is the Risk Factor Prevalence Study which consists of multi-centre surveys conducted in 1980, 1983 and 1989. These surveys were not longitudinal but were designed to provide cross-sectional estimates of risk factor levels for the Australian adult urban population at intervals. Over 22,000 Australians living in the capital cities participated in these surveys, providing a rich source of data on biomedical and behavioural risk factors.

Although there were minor differences, the three surveys used basically the same survey methods (National Heart Foundation of Australia 1982; National Heart Foundation of Australia 1985; Risk Factor Prevalence Study Management Committee 1990). A systematic probability sample of adults was selected from the latest federal electoral rolls for each capital city in Australia. Over 60% of Australians live in these capital cities and voter registration is compulsory for all Australian citizens aged 18 years and older. Core catchment areas were in Sydney (two centres), Melbourne, Brisbane, Adelaide, Perth and Hobart. Canberra and Darwin were included in the study for the first time in 1989. Persons aged 25-64 were sampled in 1980 and 1983, and persons aged 20-69 in 1989. The standard sample size was 1500 for each catchment area although some variation occurred to accommodate local requirements. For each survey, two pilot tests were conducted prior to data collection in order to assess the design and operation of the survey and to help train staff. Data collection took place between May/June and December of the survey year. The protocol for the 1989 survey was approved by the Ethics Committee of the Australian Institute of Health. Ethical clearance was not sought for the earlier surveys.

Prospective participants were mailed invitations to attend the local survey centre for a free check of risk factors for heart disease. The invitation included a specific appointment time and instructions for 12 hour fasting and other preparation for the visit. A card explaining the purpose of the (1989) survey in eleven languages was included to assist those who could not read English. The languages were Chinese, Croatian, Finnish, French, Greek, Italian,

Macedonian, Portuguese, Serbian, Spanish and Vietnamese. Visits were scheduled for mornings only in order to minimise the inconvenience of the fasting requirement. A reminder telephone call the day before the appointment was crucial to maximising response. Other approaches included reminder letters, follow-up telephone calls, home visits, opening outside normal working hours and establishing temporary clinics in areas of high non-response.

Each centre had core staff consisting of a local survey director, a clerk-receptionist and a nursing sister, and extra staff were employed by most centres as required. At the centre, participants completed a self-administered questionnaire and were then referred to a nursing sister for physical and blood pressure measurement, and blood sampling. Procedures were standardised and the promotion of uniform methods between survey centres was emphasised. Staff were trained on-site in the study protocol, procedures and measuring techniques. Adherence to the study protocol was monitored once data collection began and regular contact was maintained with each centre.

While questionnaire content evolved over the study period, the main principle was to maintain comparability between the three surveys by introducing as few changes as possible. Each survey collected information on demographic and socio-economic characteristics, physical measurements, blood pressure, blood chemistry, medical conditions and treatment, oral contraceptive use, alcohol use, smoking behaviour, dietary behaviour and exercise patterns. The appendices include a copy of the questionnaire and blood analysis form used in the 1989 survey (Appendix A) and a comparison of the data items collected in each survey (Appendix B). The front page of the questionnaire was detachable and retained at each clinic. On it, participants recorded whether they wished their results to be mailed to them, their doctor, both or neither. After blood lipid results were obtained from the central analytical laboratory, a standard results letter was posted to participants and their doctors as requested, with attention drawn to those results needing follow-up.

3.2 Response and data quality

Centres dispatched activity reports and completed questionnaires on a weekly basis to a central processing unit in Canberra. This unit handled all aspects of the registration and clerical examination of questionnaires, coding, editing and data entry, and maintained regular contact with each centre. Data quality was maintained through extensive computer validation procedures. Final response rates in each survey were very close, namely 75.9%, 75.3% and 74.5%.

Sample loss occurred for a variety of reasons including refusal to participate, incorrect address, living outside the catchment area for the duration of the survey, prison and death. Data for persons outside the specified age range and data collected through deviation from the survey protocol were excluded. Final sample numbers for each survey are given in Table 3.1. The first three columns give the number of records available for a trend analysis based on core centres and common age range.

TABLE 3.1 *Sample size for the Risk Factor Prevalence Study, centre, age, sex by year*

	1980	1983	1989 (a)	1989
Centre				
Sydney North	697	1030	810	985
Sydney South	697	867	591	697
Melbourne	681	801	700	844
Brisbane	857	1002	690	815
Adelaide	899	1051	1587	1911
Perth	900	1784	811	963
Hobart	872	1080	908	1083
Darwin	na	na	na	1000
Canberra	na	na	na	981
Age				
20-24	na	na	na	778
25-29	740	965	769	992
30-34	818	1088	805	1067
35-39	679	1146	892	1217
40-44	662	942	921	1253
45-49	658	873	739	972
50-54	757	886	662	820
55-59	737	928	626	734
60-64	552	787	683	784
65-69	na	na	na	662
Sex				
Male	2765	3740	2985	4552
Female	2838	3875	3112	4727
Total	**5603**	**7615**	**6097**	**9279**

(a)Excludes Canberra and Darwin; common age range 25-64. Compatible in scope with the 1980 and 1983 surveys.
na Not applicable.

Limitations

The study did not target Australians living in non-capital urban centres or in rural areas; no information was collected on family history of cardiovascular disease, an important risk factor; and no outcome measures are available on participants. The sampling frame (the federal electoral roll) does not include people who are ineligible to vote or who have failed to register. This may have contributed to the under-representation in the samples of migrants, the young and the more mobile.

Also, in each survey, response rates were lower in the younger and older age groups, and response rates varied between cities. The sample was also

unrepresentative with respect to persons born overseas. For example, the proportion of male respondents born overseas was close to 30% in each survey, which is less than the proportion in the target population (36% in capital city statistical divisions). Overseas-born women were also under-represented among respondents (26% cf 33%). Allowance has been made, in subsequent analyses, for imbalance in the sample structure with respect to age, city and persons born overseas by including these factors, along with survey year, in the modelling process.

The survey respondents were also unrepresentative of the target population with respect to occupation. A comparison of the sample distributions in 1980 and 1983 with estimates from the 1981 Census of Population and Housing for capital city statistical divisions shows that Professional, technical and related workers represented 22% of employed male respondents in each survey, but 15% based on census estimates for capital city statistical divisions. Equivalent figures for Administrative, executive and managerial workers were 19% and 10%. Tradesmen, production process workers and labourers were under-represented in the surveys (28% cf 38%). A similar pattern was observed for employed women. These discrepancies may reflect two factors: a tendency for survey respondents to rank their occupation higher up the socioeconomic scale, and/or a higher response rate among white collar workers. This bias needs to be kept in mind when extrapolating results from the surveys to the target population (adult Australians living in capital cities).

3.3 Measurement and definition of risk factors

Blood pressure

Two consecutive systolic and diastolic blood pressure readings were taken from the right arm, with the participant seated, using normal mercury sphygmomanometers. The readings were taken five minutes apart, and the average of the two readings was used as final value. Standard-sized cuffs (12-14 cm wide) were generally used. Large and small cuffs were available for participants who had very large or thin arms (chapter 5 contains more information).

Hypertension was defined as: treated and controlled (taking tablets for blood pressure, systolic blood pressure <160 mmHg and diastolic blood pressure <95 mmHg); treated and uncontrolled (taking tablets for blood pressure, systolic blood pressure ≥160 mmHg and/or diastolic blood pressure ≥95 mmHg); or undetected hypertension (not taking tablets for blood pressure, systolic blood pressure ≥160 mmHg and/or diastolic blood pressure ≥95 mmHg).

Blood lipids

A blood sample was taken with participants seated, after which fasting status was determined by asking whether anything had been eaten or drunk in the previous twelve hours apart from water, black tea or black coffee. The fasting requirement led to blood samples being taken in the morning. The blood specimens were analysed for plasma total cholesterol (TC), high density lipoprotein (HDL) cholesterol and triglyceride (TG) at Flinders Medical Centre, Adelaide (1980, 1983) and the Institute of Medical and Veterinary Science, Adelaide (1989). Methods are detailed in the individual survey reports (National Heart Foundation of Australia 1982; National Heart Foundation of Australia 1985; Risk Factor Prevalence Study Management Committee 1990). The central analytical laboratories satisfied the criteria for precision and accuracy as specified for standardisation by the Centres for Disease Control, Atlanta, Georgia, who provided calibration material with assigned values for cholesterol and triglyceride.

Low-density lipoprotein (LDL) cholesterol was calculated from total cholesterol, high-density lipoprotein cholesterol and triglyceride as follows: TC-HDL-TG/2.19 if TG<4.5 mmol/L[1] (The Toronto Working Group On Cholesterol Policy 1990). Trends and inequalities have also been examined for the ratio of total cholesterol to HDL cholesterol levels. A high total cholesterol value was defined as 6.5 mmol/L or greater; a high triglyceride value as 2.00 mmol/L or greater. Low HDL was defined as less than 0.91 mmol/L for men (35 mg/dl) and less than 1.16 mmol/L (45 mg/dl) for women (Helmert et al. 1990).

Height and weight

Physical measurements were taken by a nursing sister using a standard protocol. Participants were measured in socks, stockings or bare feet and light street clothing (no coats or jumpers). Height was measured to the nearest centimetre. Weight was measured to the nearest 0.1 kg (1 kg in 1980) using a metric beam balance.

Body mass index (BMI) was calculated as weight (in kilograms) divided by the square of height (in metres) after deducting 1 kg from the measured weight as an allowance for weight of clothing. For both sexes, underweight was defined as BMI<20; acceptable weight as 20≤BMI≤25; overweight as 25<BMI≤30; and obese as BMI>30.

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[1] LDL was treated as missing if TG≥4.5 mmol/L.

Smoking status

Questionnaire data were used to classify respondents as current smokers (cigarette smokers, cigar and/or pipe smokers), ex-smokers or 'never smoked regularly' and to calculate average daily consumption for current cigarette smokers.

Alcohol consumption

Respondents were asked how often they usually drank alcohol and, on a day when they drank, how many drinks they usually had. These responses were used to calculate usual drinks per week. Alcohol intake was classified as never or occasional (fewer than one drink per week), light (1-27 drinks per week for men, 1-13 drinks per week for women) or moderate to heavy (28 drinks or more per week for men, 14 drinks or more per week for women). This was consistent with National Health and Medical Research Council recommendations for 'responsible drinking behaviour' (National Health and Medical Research Council 1987b). Recent evidence suggests that modest alcohol consumption may be associated with lower cardiovascular risk (Miller et al. 1990; Jackson et al. 1991; Marmot, Brunner 1991; Rimm et al. 1991; Razay et al. 1992; Wilkins 1992; Suh et al. 1992). Accordingly, the prevalence of light alcohol intake, as defined above, has been used an indicator of reduced risk.

Dietary behaviour

The questionnaire asked `Do you add salt to your food after it is cooked?' (1983 and 1989) and `How often do you eat the fat on meat?' (1980 and 1989). For both questions the response `rarely or never' was used as an indicator of reduced risk.

Exercise during leisure-time

In the 1983 and 1989 surveys, respondents were asked about exercise taken for recreation, sport or health-fitness purposes in the previous two weeks. This included vigorous exercise (defined as causing breathlessness, puffing and panting), less vigorous exercise and walking. Vigorous exercise for three or more times a week and an average of 20 minutes or more per session was classified as aerobic exercise. No leisure-time exercise of any kind was used as an indicator of cardiovascular risk. No account was taken of exercise as part of work.

Multiple risk factors

The prevalence of individuals with two or more of the three classical risk factors for coronary heart disease was analysed using the following definitions: high blood pressure (diastolic blood pressure ≥ 95 mmHg), high blood

cholesterol (total cholesterol ≥ 6.5 mmol/L) and regular cigarette smoking (any number daily). All respondents were included whether or not they had fasted.

3.4 Data preparation and analysis

The trend analyses in chapters six and seven were based upon those variables common to the 1989 survey and the 1983 and/or 1980 surveys. The common age range was 25-64 years, and the centres common to each survey were in Sydney (two centres), Melbourne, Brisbane, Adelaide, Perth and Hobart. On this basis, survey respondents numbered 5603 in 1980, 7615 in 1983 and 6097 in 1989. The immigrant analysis in chapter four also included those respondents within the core age range from Canberra and Darwin in 1989.

The creation of a single composite file for the analyses required considerable data recoding, file manipulation, checking and testing (especially for country of birth). All analyses were conducted separately for men and women using SAS software package, version 6. Age, as at 30 June of the survey year, was calculated from date of birth provided by the respondent. Several of the chapters use analysis of covariance to compare factors levels for different population groups adjusted for age and survey design factors. Age was generally treated as four ten-year age groups to allow for non-linear relationships with risk factors (dependent variables). Distributions of all continuous variables were examined for marked departure for normality and interaction terms were considered for all models and included as appropriate. Where possible results have been given for separately for men and women, and by age groups.

Certain data were excluded from the analyses. Data from non-fasted respondents and four extreme triglyceride values (≥ 30 mmol/L) were excluded from blood lipid analyses. Data for pregnant women were excluded from all analyses which included body mass index. Procedures for measuring height in Adelaide in 1980 deviated from the study protocol and these data (and body mass index) were excluded from the analysis (weight measurements for Adelaide were included).

Missing data were negligible (<0.1%) for all survey design variables and risk factors, with the exception of body mass index (0.4%), total cholesterol (1.4%), high-density lipoprotein cholesterol (3.3%) and triglyceride (1.4%). In order to make maximum use of the information available, records containing variables with missing data were only excluded from analyses in which those variables occurred.

Chapter Four

Trends in cardiovascular risk factors in Australia

4.1 Introduction

Since the second half of the 1960s, mortality from coronary heart disease has fallen dramatically in Australia to levels which are now half the peak death rate in 1968 (Fig 4.1). Despite this, coronary heart disease remains the greatest single cause of premature death in Australia and a continuing downward trend is highly desirable. National targets to further reduce mortality from coronary heart disease are based on extrapolations of the persistent falls observed over recent decades. It is important therefore to try and explain this remarkable phenomenon so that those actions which are likely to have contributed to the decline may be continued.

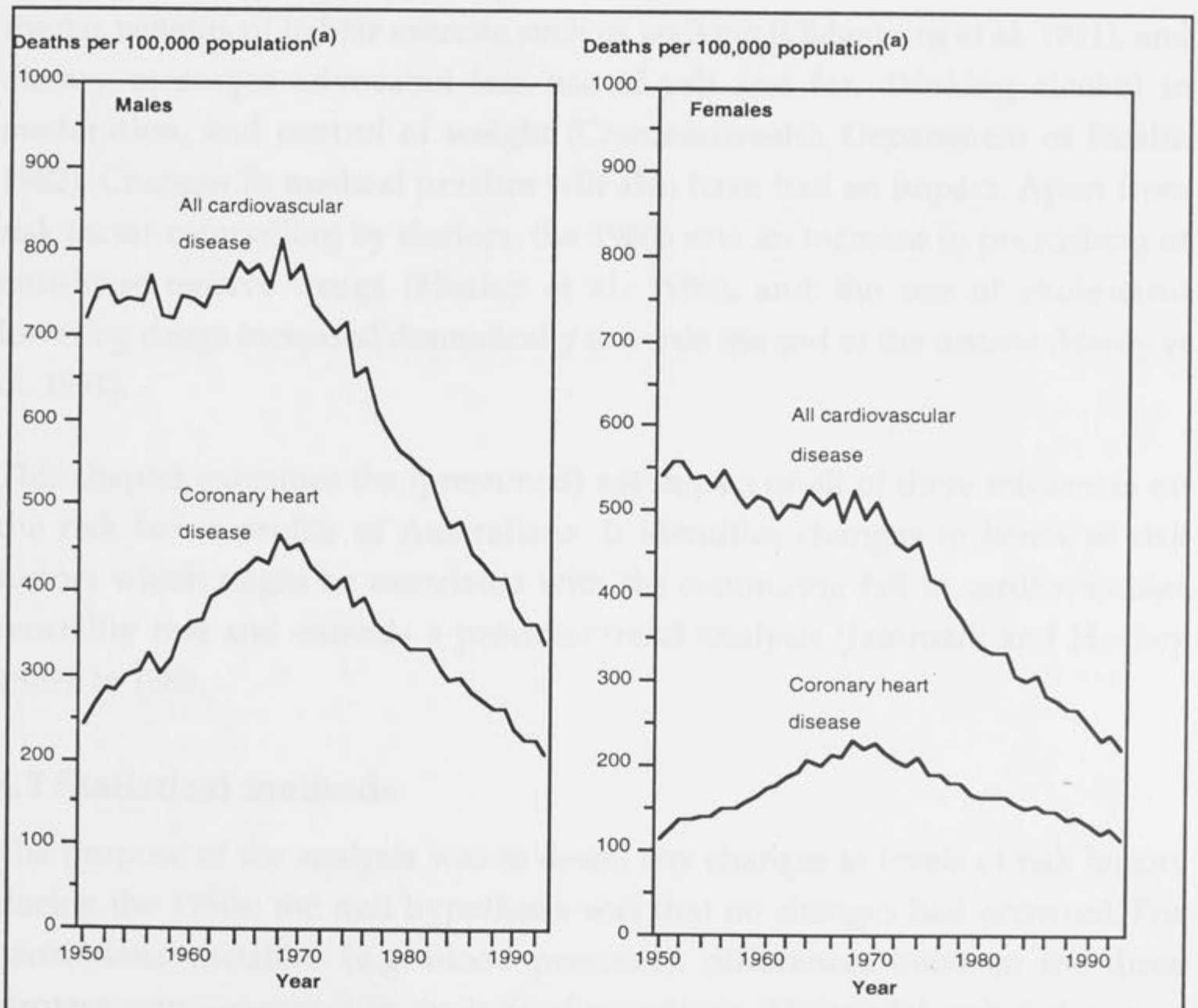

FIGURE 4.1 ***Death rates for coronary heart disease and for all cardiovascular diseases, all ages, Australia, 1950-1993***

The decline may have been caused by a reduced incidence of heart disease and/or better survival after a heart attack. The former is influenced by changes in lifestyle and levels of coronary risk factors and both factors influenced by medical intervention such as counselling, drug use, emergency care and rehabilitation. To examine the effect of these factors on the fall in mortality rate, incidence was monitored through heart attack registers (Martin et al. 1989; Dobson et al. 1988) and the prevalence of risk factors by three cross-sectional surveys commencing in 1980 (Hodge 1984). In 1984 the important measurement of medical and emergency care was added through the WHO MONICA Project (WHO MONICA Project Principal Investigators 1988).

Public education and other measures in the 1980s promoted non-smoking, regular exercise, a healthy diet, control of blood cholesterol levels, watching one's weight and drinking alcohol in moderation, all of which can affect cardiovascular risk. Non-smoking was promoted by mass media campaigns and regulatory measures (Hill et al. 1991; Powles and Gifford 1993). There was a shift in emphasis from the cardiovascular benefits of aerobic exercise to the similar benefits of lighter exercise such as walking (Oldenburg et al. 1991), and dietary messages advocated less use of salt and fat, drinking alcohol in moderation, and control of weight (Commonwealth Department of Health 1982). Changes in medical practice will also have had an impact. Apart from risk factor counselling by doctors, the 1980s saw an increase in prescribing of anti-hypertensive drugs (Hurley et al. 1990), and the use of cholesterol lowering drugs increased dramatically towards the end of the decade (Henry et al. 1991).

This chapter examines the (presumed) net impact of all of these influences on the risk factor profile of Australians. It identifies changes in levels of risk factors which might be associated with the continuing fall in cardiovascular mortality rate and extends a previous trend analysis (Jamrozik and Hockey 1989), to 1989.

4.2 Statistical methods

The purpose of the analysis was to detect any changes in levels of risk factors during the 1980s; the null hypothesis was that no changes had occurred. For continuous variables (e.g. blood pressure), differences between the three surveys were examined by analysis of covariance. The model included survey year, age (in four 10-year age groups), survey centre and birthplace (Australian-born or overseas-born) as independent variables, to allow for differences in the demographic composition of the three surveys. An

interaction term for age and year of survey was included to allow for different trends in different age groups. Use of oral contraceptives was introduced as a factor when analysing data on blood lipids for women. Multiple logistic regression was used to examine changes in categorical variables (e.g. current smoking), using the same independent variables as for continuous variables. Prevalence odds ratios and 95% confidence limits were derived from the maximum likelihood parameter estimates, using 1980 as the reference year.

4.3 Trends in risk factors

Blood pressure

During the 1980s, average systolic and diastolic blood pressures fell significantly, by 4 mmHg and 3 mmHg respectively (Tables 4.1, 4.2). Decreases were observed in men and women, and in all age groups, and for women they were more pronounced in the older age groups.

TABLE 4.1 ***Trends in blood pressure and hypertension by age, men***

	1980	1983	1989	1980-1989	
	Systolic blood pressure (mmHg)				
Age group		*Mean*		*Difference*	
25-34	126.7	126.0	123.2	-3.7#	(-5.4,-2.0)
35-44	128.9	127.9	125.3	-3.9#	(-5.6,-2.2)
45-54	136.4	136.0	132.1	-4.5#	(-6.2,-2.7)
55-64	144.6	145.1	140.6	-4.2#	(-6.0,-2.3)
25-64	*133.8*	*133.1*	*129.7*	*-4.1#*	*(-4.9,-3.2)*
	Diastolic blood pressure (mmHg)				
25-34	81.1	78.6	78.4	-2.8#	(-3.9,-1.8)
35-44	85.1	82.3	81.8	-3.4#	(-4.5,-2.3)
45-54	88.0	86.0	85.0	-3.0#	(-4.2,-1.9)
55-64	89.1	86.8	85.8	-3.4#	(-4.6,-2.3)
25-64	*85.6*	*83.2*	*82.5*	*-3.2#*	*(-3.7,-2.6)*
	Prevalence of hypertension				
Category		*Crude proportion (%)*		*Prevalence odds ratio*	
Treated and controlled	4.1	4.6	4.9	1.26	(0.97,1.63)
Treated and uncontrolled	11.5	10.7	7.3	0.60#	(0.50,0.72)
Undetected hypertension	11.1	7.0	6.6	0.57#	(0.47,0.68)
Total hypertensives	*26.7*	*22.4*	*18.8*	*0.62#*	*(0.54,0.71)*

Differences and odds ratios are adjusted for age and survey design factors, with 95% confidence limits in brackets.
* $p<0.05$, # $p<0.01$

It is difficult to standardise blood pressure measurement in repeated multi-centre surveys, and trends should usually be interpreted cautiously. However, the size of these falls cannot be explained by variation in measurement technique (see chapter 5). Falls were not experienced throughout all parts of the

distribution of blood pressure. The declines in median values were similar to those in mean values; ninetieth percentiles fell by twice as much and tenth percentiles remained essentially stationary.

TABLE 4.2 ***Trends in blood pressure and hypertension by age, women***

	1980	1983	1989	1980-1989	
	Systolic blood pressure (mmHg)				
Age group		*Mean*		*Difference*	
25-34	114.6	114.1	112.6	-2.3#	(-3.9,-0.7)
35-44	120.9	119.5	117.6	-3.6#	(-5.2,-2.0)
45-54	133.1	130.8	128.8	-4.4#	(-6.1,-2.8)
55-64	142.8	142.6	138.1	-4.8#	(-6.6,-3.1)
25-64	*127.3*	*125.9*	*123.2*	*-3.8#*	*(-4.6,-3.0)*
	Diastolic blood pressure (mmHg)				
25-34	72.9	72.2	71.8	-1.3*	(-2.2,-0.3)
35-44	78.0	76.9	75.5	-2.6#	(-3.6,-1.6)
45-54	84.4	81.7	80.8	-3.5#	(-4.5,-2.4)
55-64	86.7	83.7	82.2	-4.5#	(-5.6,-3.5)
25-64	*80.2*	*78.3*	*77.2*	*-3.0#*	*(-3.5,-2.5)*
	Prevalence of hypertension				
Category		*Crude proportion (%)*		*Prevalence odds ratio*	
Treated and controlled	6.4	6.7	7.1	1.20	(0.97,1.49)
Treated and uncontrolled	10.6	7.8	4.8	0.43#	(0.35,0.53)
Undetected hypertension	4.4	2.8	2.1	0.48#	(0.35,0.65)
Total hypertensives	*21.4*	*17.3*	*13.9*	*0.60#*	*(0.52,0.69)*

Differences and odds ratios are adjusted for age and survey design factors, with 95% confidence limits in brackets.
* $p<0.05$, # $p<0.01$

A significant reduction also occurred in the proportion of survey participants who were classified as hypertensive. This was the result of significant downward trends in the prevalence of undetected hypertension and of those receiving treatment for hypertension. Of the treated group, the prevalence of subjects with a satisfactory level of blood pressure did not change significantly. However, the prevalence of those who were not satisfactorily controlled did fall significantly. As a consequence, among people with hypertension receiving treatment, the proportion who showed a satisfactory level of blood pressure rose from 26% to 40% in men and from 38% to 60% in women. These results suggest improved detection and control of hypertension.

Falls in blood pressure similar in magnitude to those in Tables 4.1 and 4.2 were also observed when those on treatment for high blood pressure were excluded from the analysis. That is, during the 1980s, Australians not on anti-hypertensive medication (most of the adult population) experienced significant falls in blood pressure.

Blood lipids

Trends in blood lipids were different for men and women (Tables 4.3, 4.4).

TABLE 4.3 ***Trends in fasting blood lipids by age, men***

	1980	1983	1989	1980-1989	
		Total cholesterol (mmol/L)			
Age group		*Mean*		*Difference*	
25-34	5.36	5.25	5.18	-0.17#	(-0.28,-0.05)
35-44	5.66	5.62	5.68	+0.02	(-0.09,+0.14)
45-54	5.94	5.89	5.90	-0.04	(-0.16,+0.09)
55-64	5.95	5.99	5.94	-0.01	(-0.14,+0.11)
25-64	*5.72*	*5.67*	*5.66*	*-0.05*	*(-0.11,+0.01)*
		HDL cholesterol (mmol/L)			
25-34	1.25	1.27	1.19	-0.06#	(-0.09,-0.02)
35-44	1.20	1.22	1.20	0.00	(-0.03,+0.03)
45-54	1.24	1.24	1.18	-0.06#	(-0.10,-0.02)
55-64	1.25	1.25	1.19	-0.06#	(-0.10,-0.03)
25-64	*1.24*	*1.25*	*1.19*	*-0.04#*	*(-0.06,-0.03)*
		Triglyceride (mmol/L)			
25-34	1.25	1.05	1.19	-0.06	(-0.17,+0.06)
35-44	1.51	1.34	1.59	+0.09	(-0.03,+0.20)
45-54	1.62	1.41	1.68	+0.06	(-0.06,+0.18)
55-64	1.59	1.54	1.63	+0.04	(-0.08,+0.16)
25-64	*1.48*	*1.32*	*1.52*	*+0.03*	*(-0.02,+0.09)*
		LDL cholesterol (mmol/L)			
25-34	3.54	3.50	3.44	-0.09	(-0.19,+0.02)
35-44	3.78	3.83	3.78	-0.00	(-0.11,+0.10)
45-54	3.95	4.03	3.96	+0.02	(-0.09,+0.13)
55-64	3.98	4.07	4.01	+0.03	(-0.09,+0.14)
25-64	*3.80*	*3.84*	*3.79*	*-0.01*	*(-0.07,+0.04)*
		TC/HDL			
25-34	4.48	4.41	4.62	+0.14	(-0.05,+0.33)
35-44	4.94	4.91	5.06	+0.11	(-0.08,+0.30)
45-54	5.01	5.13	5.30	+0.29#	(+0.09,+0.49)
55-64	5.08	5.17	5.30	+0.21*	(+0.01,+0.42)
25-64	*4.87*	*4.88*	*5.06*	*+0.19#*	*(+0.09,+0.28)*
		Raised levels			
Category		*Crude proportion (%)*		*Prevalence odds ratio*	
TC ≥ 6.5 mmol/L	21.3	20.3	21.2	1.01	(0.86,1.18)
TG ≥ 2.0 mmol/L	20.2	13.3	20.4	1.01	(0.88,1.16)

Differences and odds ratios are adjusted for age and survey design factors, with 95% confidence limits in brackets.
* p<0.05, # p<0.01

Mean plasma total cholesterol levels decreased significantly among younger men (25-34 years) but remained the same for other ages. Men, with the exception of those aged 35-44, experienced a decrease in HDL cholesterol levels

of 5%. The net result was a significant increase in the ratio of total cholesterol to HDL cholesterol, due mainly to the increase among older men. The prevalence of high cholesterol and high triglycerides did not change significantly.

TABLE 4.4 ***Trends in fasting blood lipids by age, women***

	1980	1983	1989	1980-1989	
		Total cholesterol (mmol/L)			
Age group		*Mean*		*Difference*	
25-34	5.05	5.09	5.14	+0.09	(-0.02,+0.20)
35-44	5.32	5.29	5.21	-0.10	(-0.21,+0.01)
45-54	5.92	5.83	5.78	-0.12*	(-0.24,-0.01)
55-64	6.50	6.44	6.27	-0.23#	(-0.34,-0.11)
25-64	*5.68*	*5.63*	*5.55*	*-0.09#*	*(-0.15,-0.03)*
		HDL cholesterol (mmol/L)			
25-34	1.46	1.51	1.45	0.00	(-0.04,+0.04)
35-44	1.48	1.53	1.47	-0.01	(-0.05,+0.03)
45-54	1.53	1.57	1.52	-0.01	(-0.05,+0.03)
55-64	1.52	1.58	1.53	+0.01	(-0.03,+0.05)
25-64	*1.50*	*1.55*	*1.49*	*0.00*	*(-0.02,+0.02)*
		Triglyceride (mmol/L)			
25-34	0.87	0.79	0.95	+0.07*	(+0.01,+0.14)
35-44	0.89	0.84	0.98	+0.09#	(+0.03,+0.16)
45-54	1.10	1.02	1.17	+0.07*	(+0.01,+0.14)
55-64	1.34	1.24	1.42	+0.09*	(+0.02,+0.16)
25-64	*1.04*	*0.96*	*1.11*	*+0.08#*	*(+0.05,+0.11)*
		LDL cholesterol (mmol/L)			
25-34	3.19	3.23	3.25	+0.07	(-0.04,+0.17)
35-44	3.44	3.38	3.30	-0.14#	(-0.24, 0.04)
45-54	3.89	3.78	3.73	-0.15#	(-0.26,-0.05)
55-64	4.37	4.29	4.08	-0.28#	(-0.39,-0.17)
25-64	*3.71*	*3.64*	*3.55*	*-0.12#*	*(-0.18,-0.07)*
		TC/HDL			
25-34	3.63	3.53	3.71	+0.09	(-0.05,+0.23)
35-44	3.80	3.65	3.74	-0.05	(-0.19,+0.09)
45-54	4.09	3.96	4.05	-0.04	(-0.18,+0.11)
55-64	4.53	4.36	4.39	-0.13	(-0.28,+0.03)
25-64	*4.00*	*3.85*	*3.94*	*-0.03*	*(-0.10,+0.04)*
		Raised levels			
Category		*Crude proportion (%)*		*Prevalence odds ratio*	
TC ≥ 6.5 mmol/L	21.8	19.8	19.5	0.92	(0.80,1.07)
TG ≥ 2.0 mmol/L	7.0	5.5	8.1	1.28*	(1.03,1.51)

Differences and odds ratios are adjusted for age and survey design factors, with 95% confidence limits in brackets.
* $p<0.05$, # $p<0.01$

Among women, mean plasma cholesterol fell in the older age groups. Mean HDL cholesterol in 1989 was similar to that in 1980 but the level in 1983 was statistically significantly higher ($p<0.0001$), for reasons unknown. Mean triglyceride level and the prevalence of high triglycerides increased significantly between 1980 and 1989. LDL cholesterol levels decreased significantly in women aged 35 years or older.

Mean triglyceride levels were significantly lower in 1983 than in 1980 or 1989, for both men and women. The reason is unclear as blood sampling and measurement followed strict guidelines. Analysis of median values indicates that the lower 1983 levels were not the result of a few extremely high values in 1980 and 1989.

There were significant increases in the prevalence of men and women receiving treatment to lower blood fat, from 1.6% to 3.1% for men (odds ratio 2.2; $p<.0001$), and from 1.1% to 2.1% for women (odds ratio 2.2; $p<.001$). Among women aged 55-64, prevalence increased from 2.0% to 6.3% (odds ratio 3.4; $p<0.001$).

Overall, the results show no clear time trend in the lipid profiles of Australian men and women. The only beneficial shifts were decreases in average plasma total cholesterol by 3% in men aged 25-34, and by a similar amount in older women (45-64), among whom there was also a significant increase in the prevalence of treatment to lower blood cholesterol level. Average triglyceride levels increased in women by 8% with a consequent increase in hypertriglyceridemia.

Weight for height

There was an increase in average weight which was greater among women than men (Tables 4.5, 4.6). Age specific increases in weight in men (less than those observed in women) resulted in increases of 13% in odds of being overweight and 24% in being obese. There was no weight increase during the 1980s among men aged 35-44 but this age group was the heaviest in 1980. Women in 1989 were over 3 kg heavier on average than their 1980 counterparts. An increase in average weight occurred for all ages, with consequent increases in body mass index (average height did not change). The odds of being overweight increased by 32%, and odds of being obese by 70%.

The overall picture is one of increasing weight of Australian women across the whole of the weight distribution range, with consequent increases in

prevalence of those overweight or obese and a decrease in the prevalence of those underweight. Men experienced a smaller increase in average weight than women, but the prevalence of overweight men (44%) remained considerably greater than that for women (25%).

TABLE 4.5 Trends in weight, and weight for height by age, men

	1980	1983	1989	1980-1989	
		Weight (kg)			
Age group		*Mean*		*Difference*	
25-34	75.6	75.1	77.5	+1.7#	(+0.4,+2.9)
35-44	78.8	77.2	79.0	-0.1	(-1.3,+1.2)
45-54	77.4	78.8	80.4	+2.9#	(+1.6,+4.2)
55-64	76.4	76.9	78.6	+2.2#	(+0.9,+3.5)
25-64	*77.0*	*77.0*	*78.8*	*+1.7#*	*(+1.0,+2.3)*
		Body mass index (kg/m^2)			
25-34	24.2	24.2	24.9	+0.6#	(+0.2,+1.0)
35-44	25.5	25.2	25.5	-0.1	(-0.5,+0.3)
45-54	25.8	26.0	26.4	+0.5#	(+0.1,+0.9)
55-64	25.8	25.9	26.3	+0.4*	(+0.0,+0.9)
25-64	*25.3*	*25.3*	*25.7*	*+0.4#*	*(+0.2,+0.6)*
		Weight for height			
Category		*Crude proportion (%)*		*Prevalence odds ratio*	
Underweight	4.8	4.7	3.1	0.72*	(0.53,0.96)
Acceptable	45.4	46.2	41.3	0.86#	(0.76,0.96)
Overweight	40.6	40.0	44.1	1.13*	(1.01,1.27)
Obese	9.3	9.1	11.5	1.24*	(1.02,1.50)
Overweight or obese	49.8	49.1	55.6	1.23#	(1.09,1.38)

Differences and odds ratios are adjusted for age and survey design factors, with 95% confidence limits in brackets.
* $p<0.05$, # $p<0.01$

An increasing body mass index in men and in women was also seen during the 1980s in the United Kingdom, the United States (Gregory et al. 1990; Sprafka et al. 1990; Kuczmarski 1992) and, to a lesser extent, in Sweden (Kuskowska-Wolk and Bergström 1993a; 1993b). Trends for New Zealand (Jackson et al. 1990), Italy (Research Group of the Italian National Research Council 1987), and Finland (Jalkanen et al. 1989) were quite different to those in Australia. It is evident that public health advice in Australia to maintain a healthy body weight (National Health and Medical Research Council 1992a) was unsuccessful and that national targets to reduce the prevalence of overweight and obese adults are unlikely to be achieved by the year 2000.

TABLE 4.6 *Trends in weight, and weight for height by age, women (a)*

	1980	1983	1989	1980-1989	
		Weight (kg)			
Age group		*Mean*		*Difference*	
25-34	58.7	60.4	61.8	+2.8#	(+1.6,+4.1)
35-44	61.4	62.5	63.9	+2.3#	(+1.1,+3.5)
45-54	63.2	64.8	67.3	+4.0#	(+2.8,+5.3)
55-64	63.7	64.6	67.0	+3.2#	(+1.9,+4.5)
25-64	*61.7*	*63.0*	*64.8*	*+3.1#*	*(+2.5,+3.7)*
		Body mass index (kg/m^2)			
25-34	22.1	22.8	23.2	+0.9#	(+0.4,+1.4)
35-44	23.3	23.7	24.2	+0.8#	(+0.3,+1.2)
45-54	24.2	25.1	25.7	+1.3#	(+0.8,+1.8)
55-64	25.0	25.4	26.2	+1.0#	(+0.5,+1.5)
25-64	*23.7*	*24.1*	*24.7*	*+1.0#*	*(+0.7,+1.3)*
		Weight for height			
Category		*Crude proportion (%)*		*Prevalence odds ratio*	
Underweight	16.7	14.1	11.8	0.69#	(0.59,0.82)
Acceptable	55.0	53.4	49.8	0.81#	(0.72,0.91)
Overweight	20.2	22.0	25.1	1.32#	(1.15,1.51)
Obese	8.0	10.5	13.2	1.70#	(1.40,2.06)
Overweight or obese	28.3	32.5	38.3	1.58#	(1.39,1.79)

Differences and odds ratios are adjusted for age and survey design factors, with 95% confidence limits in brackets.
* p<0.05, # p<0.01 (a) excludes pregnant women

Smoking

Smoking prevalence declined significantly in men and women, while the proportion of ex-smokers increased (Tables 4.7, 4.8). The proportion of `never smokers' increased for men but remained the same for women. The data on smoking are self-reported, unvalidated (biochemically), and subject to under-reporting. Nevertheless, the marked decline observed in smoking prevalence is consistent with declines observed in other Australian studies (Australian Bureau of Statistics 1992; Hill et al. 1991) and in tobacco consumption since the mid 1970s (National Campaign Against Drug Abuse 1992).

The data suggest that cessation of smoking and a decreased rate of recruitment both contributed to the fall in men, while smoking cessation was more important in women. An age-specific analysis (not shown) reveals a decline in each 10-year age group for men and women. These results may foreshadow a change in the trend for mortality from lung cancer for women which is currently increasing at 3.0% per annum, compared with a decreasing trend of 1.5% per annum among men (Bennett et al. 1994).

TABLE 4.7 ***Trends in smoking and alcohol consumption by age, men***

	1980	1983	1989	1980-1989	
		Smoking status			
Category		*Crude proportion (%)*		*Prevalence odds ratio*	
Current smokers	37.5	34.8	26.2	0.60#	(0.53,0.67)
Ex-smokers	28.6	30.1	34.2	1.31#	(1.17,1.47)
Never smoked	33.9	35.2	39.5	1.28#	(1.14,1.42)
Cigarette smokers	34.3	32.3	25.0	0.64#	(0.57,0.72)
		Daily cigarette consumption			
		Mean		*Difference*	
Cigarettes	20.3	19.6	19.3	-0.8	(-2.1,0.4)
		Alcohol consumption			
Category		*Crude proportion (%)*		*Prevalence odds ratio*	
Never/occasional	8.8	11.9	12.4	1.47#	(1.24,1.75)
Light	78.4	77.9	80.7	1.12	(0.99,1.28)
Moderate/ heavy	12.8	10.2	6.9	0.53#	(0.44,0.63)

Differences and odds ratios are adjusted for age and survey design factors, with 95% confidence limits in brackets.
* p<0.05, # p<0.01 n.a not available.

TABLE 4.8 ***Trends in smoking and alcohol consumption by age, women***

	1980	1983	1989	1980-1989	
		Smoking status			
Category		*Crude proportion (%)*		*Prevalence odds ratio*	
Current smokers	26.9	24.5	20.9	0.71#	(0.63,0.81)
Ex-smokers	15.1	16.6	20.1	1.42#	(1.24,1.63)
Never smoked	58.0	58.9	59.0	1.04	(0.94,1.16)
Cigarette smokers	26.8	24.4	20.8	0.71#	(0.63,0.81)
		Daily cigarette consumption			
		Mean		*Difference*	
Cigarettes	15.6	16.1	16.5	1.4*	(0.3,2.5)
		Alcohol consumption			
Category		*Crude proportion (%)*		*Prevalence odds ratio*	
Never/occasional	19.4	24.6	25.4	1.48#	(1.30,1.68)
Light	74.8	69.9	70.8	0.78#	(0.70,0.88)
Moderate/ heavy	5.7	5.5	3.8	0.66#	(0.52,0.84)

Differences and odds ratios are adjusted for age and survey design factors, with 95% confidence limits in brackets.
* p<0.05, # p<0.01 n.a not available.

Although smoking prevalence declined markedly, changes in the daily consumption of manufactured cigarettes by smokers were relatively small (although marginally statistically significant in women). A similar finding has been reported for Australian schoolchildren (Hill et al. 1990). If those who quit were lighter than average smokers then there must have been a compensating decline in consumption among continuing smokers. Alternatively, if those who quit were drawn evenly from across the distribution of consumption, then the

quit smoking message had little effect on consumption among those who continued.

Alcohol consumption

The self-reported data on alcohol consumption suggest that Australians have become more responsible in their use of alcohol. The past decade saw a reduced prevalence of men and women classified as moderate to heavy drinkers and an increase in those classified as abstainers or occasional drinkers (Tables 4.7, 4.8). This is consistent with the downward trend in the apparent per capita consumption of alcohol during the 1980s (National Campaign Against Drug Abuse 1992). Recent evidence links modest alcohol consumption with lower cardiovascular risk (Marmot and Brunner 1991; Jackson et al. 1991). If this is so, then the trend has not improved the overall cardiovascular risk profile of most Australian adults as the prevalence of light drinkers did not increase. The reduction in the upper extreme of the consumption distribution should have beneficial outcomes through decreased risk of coronary heart disease, ischaemic and haemorrhagic stroke, and hypertension.

Dietary behaviour

The marked positive trends in self-reported dietary behaviour (Tables 4.9, 4.10) suggest that the messages of the 1980s - to use less salt and avoid eating too much fat (Commonwealth Department of Health 1982) - were effective, although the differential in favour of women remains.

TABLE 4.9 *Trends in dietary behaviour, exercise during leisure time and multiple risk factors by age, men*

	1980	1983	1989	1980-1989	
Dietary behaviour					
Do not add salt	n.a	30.9	47.9	2.08#	(1.88,2.31)
Do not eat fat on meat	41.2	n.a	48.3	1.35#	(1.21,1.50)
Exercise during leisure-time					
Aerobic	n.a	9.3	9.0	0.99	(0.83,1.16)
Vigorous	n.a	31.5	31.9	1.02	(0.91,1.15)
Less vigorous	n.a	27.1	30.2	1.17#	(1.05,1.31)
Walking	n.a	47.4	53.9	1.28#	(1.17,1.40)
No exercise	n.a	32.5	28.5	0.83#	(0.74,0.92)
Multiple risk factors					
2 or 3 risk factors	15.6	12.6	10.3	0.62#	(0.53,0.73)

Differences and odds ratios are adjusted for age and survey design factors, with 95% confidence limits in brackets. Some comparisons are for 1983-1989. * $p<0.05$, # $p<0.01$ n.a not available.

The aggregate figures indicate a sharp reduction in the proportion of people who add salt to food after it is cooked, although additional analysis shows that older people have not responded as enthusiastically to this message as younger

people. Neither indicator was intended to represent a measure of intake as, for example, 75% of salt in the diet comes from manufactured food. However, it is assumed that these indicators are reasonable proxies for a range of dietary components, and as such can be seen as broad indicators of healthy dietary behaviour.

Exercise during leisure time

Walking for recreation or exercise and other forms of less vigorous exercise became significantly more popular, while the prevalence of aerobic exercise and vigorous exercise remained unchanged (Tables 4.9, 4.10). Overall, the results lend little support to the popular view that recreational exercise patterns have improved markedly during recent years. Together with other Australian data (Bauman et al. 1990), they suggest a modest increase in participation in less vigorous forms of exercise, including walking, but no change in the prevalence of vigorous exercise. The net effect was a marginal reduction in the proportion who reported being totally sedentary during leisure time.

TABLE 4.10 *Trends in dietary behaviour, exercise during leisure time and multiple risk factors by age, women*

	1980	1983	1989	1980-1989	
Dietary behaviour					
Do not add salt	n.a	41.6	56.7	1.79#	(1.62,1.97)
Do not eat fat on meat	50.1	n.a	63.4	1.76#	(1.59,1.96)
Exercise during leisure-time					
Aerobic	n.a	5.1	5.2	1.02	(0.83,1.27)
Vigorous	n.a	25.0	25.6	1.03	(0.92,1.16)
Less vigorous	n.a	22.9	26.2	1.21#	(1.08,1.35)
Walking	n.a	54.1	59.8	1.25#	(1.13,1.38)
No exercise	n.a	31.6	29.3	0.90*	(0.81,1.00)
Multiple risk factors					
2 or 3 risk factors	10.9	8.0	6.6	0.61#	(0.50,0.74)

Differences and odds ratios are adjusted for age and survey design factors, with 95% confidence limits in brackets. Some comparisons are for 1983-1989. * $p<0.05$, # $p<0.01$ n.a not available.

There has not been a marked increase in aerobic activity, but, to the extent that health benefits accrue from less vigorous forms of recreational exercise, the overall trend is encouraging. Evidence suggests that both leisure-time activity and exercise at work (not measured) reduce coronary risk, although their relative contribution is unclear (Salonen et al. 1988).

Multiple risk factors

Between 1980 and 1989, the prevalence of individuals with two or more risk factors (high blood pressure, cigarette smoking, high total cholesterol) fell from

16% to 10% in men and from 11% to 7% in women, primarily in the three older age groups. A fall in the simultaneous occurrence of high blood pressure and cigarette smoking was important for men and women. Among women aged 55-64, there was a marked decrease in the proportion who had both high blood pressure and high total cholesterol, from 11% to 3%. The simultaneous occurrence of all three major risk factors within the one individual was rare (around 1%).

4.4 Discussion

Australia is one of few countries to have conducted three repeat surveys of cardiovascular risk factors and these results help to redress the international paucity of data on trends in risk factors (Epstein 1989; Pearson and Pyorala 1989; Beaglehole 1990; Winkleby 1994). Although the scope of the study is limited to Australians aged 25-64, living in the capital cities and physically able to attend a clinic, the surveys represent the only nationwide data collections in Australia which include physical measurements and blood sampling.

In this analysis, consistency of methods and accuracy are most important, as trends in risk factors over time are likely to be relatively small. Observer error and non-response bias are both potential confounders in such an analysis. The steps taken to minimise their effects were described in the individual survey reports. Non-respondents in surveys such as these tend to be of lower socioeconomic status and to have worse risk factor profiles than respondents (Criqui et al. 1978; Bergstrand et al. 1983), but characteristics of non-respondents are relatively stable over time (Sprafka et al. 1990). No data were collected on the characteristics of non-respondents in the Risk Factor Prevalence Study, but the consistency in overall response rates over time offers some assurance against biased trend estimates.

Although repeated cross-sectional surveys have limited ability to investigate relationships between risk factors, some points can be made. The fall in blood pressure across all age groups is consistent with observed changes in dietary behaviour (less added salt, fat on meat and alcohol) and occurred while body fatness was increasing. It might be speculated that the decrease in smoking prevalence could explain the increase in body mass index, however separate analysis of these data does not support this (Boyle et al. 1994). Increasing treatment to lower blood cholesterol levels may have contributed to the fall in total cholesterol among older women. Falls in the prevalence of multiple risk factors in both men and women are especially important because interaction between factors increases risk more than additively (Manson et al. 1992a).

The marked decline in smoking has been attributed to the public response to smoking reduction programs, including mass media campaigns, individual efforts of doctors, other health professionals and teachers, legislative and regulative changes, and price increases (Hill et al. 1991). The experience in Victoria, of a phased ban on advertising and use of taxes from tobacco sales to replace tobacco sponsorship of sports and arts and to fund health promotion, has been used as an example of how coordinated legislative and voluntary sector action can have a substantial impact on public behaviour (Powles and Gifford 1993). The groups which have maintained a higher prevalence of smoking are adults aged 20-29 (Hill et al. 1991), Aboriginal and Torres Strait Islanders (Australian Bureau of Statistics 1992), people without a partner (National Health Strategy 1992), and those of lower socioeconomic status as measured by educational attainment (Hill et al. 1991; National Health Strategy 1992), occupation (Australian Bureau of Statistics 1992; Hill et al. 1991), area of residence and income (National Health Strategy 1992). Trends in smoking prevalence by occupation group and by educational attainment are discussed in Chapters 7 and 8 respectively.

During 1981-1992, mortality from coronary heart disease in Australia fell by 3.6% per annum in men and 2.6% per annum in women (Bennett et al. 1994). These declines are the net effect of changes in incidence and case fatality, whose determinants include risk factor levels (Dobson 1987) and medical care (Czarn et al. 1992). Changes in diet, smoking, hypertension indices and medical care are all consistent with the trends in coronary heart disease mortality rates (Thomson et al. 1988; Al-Roomi et al. 1989). Mortality from stroke is currently falling at 4.6% per annum (Bennett et al. 1994), consistent with the significant fall in mean blood pressure observed in this study and the short lag time between lowering of blood pressure and prevention of fatal strokes (Collins et al. 1990).

To conclude, with the exception of body fatness and blood lipids, trends in population risk factor levels are generally consistent with falling cardiovascular mortality rates. Declines were especially marked for cigarette smoking and blood pressure. These two risk factors have short lead times between their modification and subsequent effect on mortality from coronary heart disease and stroke (Collins et al. 1990; Manson et al. 1992a) and are likely to have contributed to the falls in cardiovascular mortality observed in Australia. Diet has a significant impact on cardiovascular risk factors and disease (Shrapnel et al. 1992) and this analysis found favourable trends in dietary behaviour. Patterns of exercise during leisure time have changed little. The secular fall in mortality from coronary heart disease has been sustained while body fatness

increased. If this trend in body fatness continues it will counteract the improvements in several other risk factors and ultimately in cardiovascular morbidity and mortality.

Chapter Five

The effect of blood pressure measurement error on cross-sectional and trend analysis

5.1 Introduction

Blood pressure is especially liable to measurement error, dependent as it is upon observer skill in objective and accurate reading and recording of data. This chapter estimates the extent to which the significant falls in blood pressure reported in chapter 4 may have been influenced by measurement error between survey centres over time, and by variation in measurement technique. The results have more general applicability as blood pressure is commonly measured in population studies of cardiovascular diseases; the use of standard mercury sphygmomanometers is still popular; and blood pressure determination frequently consists of two consecutive readings recorded to the nearest 2 mmHg (WHO MONICA Project et al. 1989). Data from such studies can be used to make regional comparisons in blood pressure levels and hypertension prevalence, and repeat studies can be used to estimate trends over time. There are few published data on secular trends in blood pressure (Beaglehole 1990) but this situation should change as the number of countries with repeat surveys increases. The magnitude of secular differences is likely to be relatively small and hence especially sensitive to differential measurement error. The question then arises as to whether findings have been unduly influenced by measurement error or variation in measurement technique.

Two common manifestations of measurement error in epidemiological studies are last digit preference for zero and the proportion of identical readings. Preference for zero as the last digit when recording blood pressure using normal mercury sphygmomanometers is well documented (Rose et al. 1964; Hessel 1986; Choi et al. 1978; Eilersten and Humerfelt 1968; Hypertension Detection and Follow-up Program Cooperative Group 1978). It was observed in NHANES I and II (National Center for Health Statistics et al. 1986) and continues to be a common phenomenon (Hense et al. 1990). It effects the shape of the distribution curve (Hense et al. 1990) and reduces the power of statistical tests, thereby making it more difficult to assess associations between blood pressure and other potential risk factors (Hessel 1986). The use of the proportion of identical duplicate measurements as an indicator of measurement technique and data quality is less well documented. High

prevalence may cause a shift in the entire blood pressure distribution (Hense et al. 1990).

This chapter uses data from the Risk Factor Prevalence Study to estimate the effect of these measurement characteristics on cross-sectional and trend analyses. The three cross-sectional surveys each comprised seven core centres, providing data for 21 independent populations for the analysis. In common with other repeat cross-sectional surveys underway in other countries, the ability of the Australian study to detect geographical or temporal changes in blood pressure characteristics of the population will be impaired by variation in measurement technique between centres and between surveys.

5.2 Criteria for assessing data quality

The quality of blood pressure measurement has been assessed in terms of deviation from the study protocol and variation in measurement technique between centres and surveys. Since the protocol specifies measurement to the nearest even 2 mmHg, it follows that departure from the protocol is indicated by deviation from a uniform frequency distribution of even last digits and the occurrence of odd last digits. As there was very little last digit preference for any even digit apart from zero, its prevalence has been used as the indicator of departure from the protocol rather than a composite statistic based on the frequency of all even digits (Hense et al. 1990). Variation in measurement technique has been examined by comparing the two consecutive measurements of blood pressure. The set of indicators includes the proportion of identical duplicate measurements together with other statistics which have also been advocated as important indicators of measurement quality (Canner et al. 1991). Unlike departure from the study protocol, there are no gold standards against which variation in measurement technique may be compared. The total battery of indicators used to examine the quality of the Australian data is as follows.

Deviation from study protocol

- last digit preference (for zero)
- proportion of odd readings (odd final digit)

Variation in measurement technique

- proportion of identical duplicate readings
- proportion of second readings exceeding first readings
- mean absolute difference between readings
- mean (signed) difference between first and second readings

Blood pressure measurements have been assessed for 19,315 respondents from seven independent centres which participated in each of the 1980, 1983 and 1989 Australian risk factor prevalence surveys. All readings were taken by nursing sisters using normal mercury sphygmomanometers. Most centres used one sphygmomanometer (the maximum was 3) during the data collection period and employed one or two nursing sisters (although one centre used 6 observers).

All nursing sisters received prior on-site training in blood pressure measurement according to the standard procedure (Box 5.1) which followed the WHO MONICA protocol (World Health Organization 1986). The Prineas tapes and training program were used. The final systolic and diastolic blood pressure readings were the average of two successive readings taken five minutes apart. Almost all physical examinations took place during the morning, because of lipid fasting requirements, and this controlled for any time-of-day effect on blood pressure (Padfield et al. 1990; Muller and Tofler 1991; Heller et al. 1978). Blood pressure measurements were taken after completion of a questionnaire, and after the anthropometric measurements. This gave respondents time to become familiar with their surroundings.

Box 5.1 Procedure for measuring blood pressure in the National Heart Foundation Risk Factor Prevalence Study.

- seat participant with right arm resting on table and with inner elbow level with the heart
- ensure that sphygmomanometer scale is facing the nursing sister (but not the participant) with middle of mercury scale level with eyes and close enough to read mercury column
- check cuff size is appropriate
- find the brachial artery at the elbow
- centre the cuff bladder over the artery with the lower end of the cuff about 2 cm above the elbow band
- while feeling the pulse, inflate the cuff until the pulse disappears (palpated systolic pressure)
- rapidly deflate the cuff
- wait 30 seconds
- rapidly inflate to 30 mmHg above the palpated systolic pressure noted before
- release air in the cuff slowly at a rate of 2 mmHg (one gauge interval) per second
- note level of first clear tapping sound to nearest 2 mmHg, closest to the upper edge of the mercury meniscus. There should be no odd-number readings.
- record the above as systolic
- lower the pressure further and record the point where all sounds disappear. Record this point as the diastolic phase 5. (Do not record the earlier point where muffling begins)
- wait 5 minutes
- repeat measurement and record as above

Measurements were incomplete for seven of the 19,315 participants; that is, at least one of the first and second readings for systolic and diastolic blood pressure was missing. These observations were excluded from the analysis. A faulty sphygmomanometer resulted in high blood pressure readings in Melbourne in 1980. This was detected at an early stage of the data collection and the faulty observations for 154 men and 165 women were adjusted (National Heart Foundation of Australia 1982). Since the original observations could not be reconstructed these data were excluded from all calculations of last digit preference for zero and proportion of odd readings.

5.3 Results

Departure from the study protocol

Bias towards a last digit of zero was greater in the first survey, reaching 90% in one centre (Table 5.1). This was far in excess of the 20% expected if each even digit were equally likely. Only in one centre was the preference for zero consistently 30% or less.

TABLE 5.1. *Last digit preference for zero (a), by centre and survey*

	Systolic blood pressure			Diastolic blood pressure		
Centre	1980	1983	1989	1980	1983	1989
			Per cent			
Sydney North	34	22	24	29	24	25
Sydney South	51	33	36	52	35	34
Melbourne	37	34	19	31	48	19
Brisbane	89	21	20	92	21	21
Adelaide	59	24	33	51	23	34
Perth	29	21	18	30	23	25
Hobart	45	36	17	44	29	20
All centres	50	27	25	49	28	26

(a) First and second readings combined.

The likely effect of gross last digit preference for zero on blood pressure distributions was estimated by rounding all blood pressure readings to the nearest 10 mmHg for those survey centres in Table 5.1 with a last digit preference for zero of less than 40%. As expected, the simulation demonstrated that preference for zeros has no appreciable effect on the means and standard deviations. After averaging the first and second readings, the average change in mean systolic and diastolic pressure based on rounded rather than observed data was less than +0.1 mmHg, and standard deviations increased by less than 0.2 mmHg. The simulation assumed, of course, that there was no systematic rounding up or rounding down of the true readings by the observer. However, with this assumption, gross digit preference is likely to have an effect on the

estimate of the proportion at risk. Pressures immediately below the cut-off point will have been rounded up, thus increasing the proportion counted in the upper category. For example, in men, the prevalence of high systolic blood pressure (160 mmHg or above) increased on average from 7.8% before the simulation to 9.2% after, and from 5.5% to 6.4% in women. This represents an increase of 17% in the magnitude of the estimate at risk for both men and women.

In each survey, preference for zero was concentrated more towards the lower end of the blood pressure range (Table 5.2) which suggests that observers may have considered low values to be less important and therefore exercised less care, or that they experienced more difficulty with very low readings.

TABLE 5.2 *Last digit preference for zero (a), by level of blood pressure and survey*

Level (mmHg)	1980	1983	1989
		Per cent	
Systolic blood pressure			
110 or less	63.8	36.8	36.0
111-120	54.2	28.0	24.0
121-130	47.1	24.0	21.4
131-140	42.7	22.7	20.7
141-150	44.9	22.3	19.9
151-160	48.2	24.3	22.5
161 or more	46.2	23.4	24.1
Diastolic blood pressure			
70 or less	65.0	41.4	41.0
71-80	52.2	29.6	26.3
81-90	44.3	21.4	19.2
91-100	33.9	16.2	18.3
101 or more	37.8	20.5	17.5

(a) First and second readings combined

Other studies have found higher digit preference at both extremes of the pressure range (Hessel 1986; Rose et al. 1964). One possible explanation is that observers would be taken unawares by extremely high or low values and therefore more likely to round (Rose et al. 1964). It has also been suggested that the pattern results from a tendency to measure blood pressure more precisely near diagnostically important points (Hessel 1986).

Any occurrence of readings with an odd digit also represents deviation from the study protocol. Generally, this was well controlled (less than 2%) but there were a few exceptions (39% in Adelaide in 1980, 11% in Perth in 1980 and 15% in Adelaide in 1983). Occurrence was similar for systolic and diastolic measurements, and the most common final odd digit was '5'. The occurrence of odd digits is likely to reflect forgetfulness of the protocol or a desire for extra

accuracy by the observer. It can also indicate a loss of accuracy. In Adelaide in 1980, over 90% of readings ended in '0' or '5', which suggests that measurements were made to the nearest 5 mmHg rather than 2 mmHg.

Variation in measurement technique

The indicators of measurement technique (Tables 5.3 to 5.6) are not mutually independent, but they do serve to illustrate that most centres showed marked differences in recording practice between surveys. The proportion of identical duplicate measurements was greater in 1980 than that observed in the later surveys (Table 5.3), and it varied markedly between centres within surveys and between surveys for the same centre. Studies invariably report that average blood pressure falls between successive readings, but there are exceptions (Canner et al. 1991). Table 5.4 demonstrates the frequency with which measured blood pressure increased between successive readings in a population survey setting and shows the variation between survey centres. Both absolute and signed mean differences (Tables 5.5 and 5.6) have been advocated as indicators of observer variation (Canner et al. 1991).

TABLE 5.3 *Proportion of identical duplicate readings, by centre and survey*

	Systolic blood pressure			Diastolic blood pressure		
Centre	1980	1983	1989	1980	1983	1989
			Per cent			
Sydney North	23	9	13	30	15	23
Sydney South	43	25	27	50	34	35
Melbourne	42	9	8	55	25	14
Brisbane	37	12	8	57	17	16
Adelaide	61	18	12	67	35	21
Perth	9	4	17	16	24	17
Hobart	43	3	39	56	3	46
All centres	37	10	17	48	22	25

TABLE 5.4 *Second reading exceeded first reading, by centre and survey*

	Systolic blood pressure			Diastolic blood pressure		
Centre	1980	1983	1989	1980	1983	1989
			Per cent			
Sydney North	28	21	18	29	38	28
Sydney South	22	21	11	24	26	9
Melbourne	0	27	22	1	32	32
Brisbane	28	26	28	23	41	41
Adelaide	2	12	21	1	11	25
Perth	12	14	32	17	19	54
Hobart	20	23	23	15	46	24
All centres	16	20	22	15	29	30

TABLE 5.5 *Mean absolute difference between first and second readings, by centre and survey*

Centre	Systolic blood pressure			Diastolic blood pressure		
	1980	1983	1989	1980	1983	1989
	mmHg					
Sydney North	4.2	6.6	3.9	2.8	4.3	2.7
Sydney South	2.2	4.7	5.9	1.8	2.8	3.8
Melbourne	3.0	7.7	7.7	1.8	3.9	4.9
Brisbane	5.8	6.5	7.1	3.6	3.7	4.5
Adelaide	2.3	5.0	7.1	1.6	2.4	4.2
Perth	6.1	6.3	4.7	3.5	2.4	4.0
Hobart	2.5	5.3	2.8	1.5	4.6	2.0
All centres	3.8	6.0	5.7	2.4	3.4	3.7

TABLE 5.6 *Mean difference between first and second readings (a), by centre and survey*

Centre	Systolic blood pressure			Diastolic blood pressure		
	1980	1983	1989	1980	1983	1989
	mmHg					
Sydney North	1.5	4.5	2.5	0.6	0.7	0.9
Sydney South	0.4	2.3	4.7	-0.0	0.7	2.7
Melbourne	3.0	4.0	5.1	1.8	0.6	1.8
Brisbane	0.9	3.8	3.5	-0.2	0.3	0.2
Adelaide	2.0	3.9	4.7	1.5	1.9	1.6
Perth	4.8	5.3	1.4	2.5	1.4	-1.6
Hobart	0.9	2.6	1.0	0.5	0.5	0.4
All centres	2.0	3.9	3.3	1.0	0.9	0.9

(a) First reading minus second reading.

Further analysis showed that, for systolic blood pressure, the prevalence of identical duplicate measurements (Table 5.3) was significantly and inversely correlated with the mean difference between the duplicate measurements (Table 5.6). That is, centres with a high prevalence of identical duplicate measurements tended to have a smaller average decrease between first and second readings, as would be expected. The average fall in systolic level between first and second readings for centres with low prevalence of identical duplicate measurements (less than 10%) was 4.3 mmHg. The average fall for centres with a high prevalence (greater than 30%) was 1.4 mmHg. This relationship was modelled in order to estimate the effect of differential prevalence of identical duplicate readings on comparisons between centres or surveys. The scatter of points, combined with the theoretical endpoint (1,0), suggested using the exponential function (Figure 5.1).

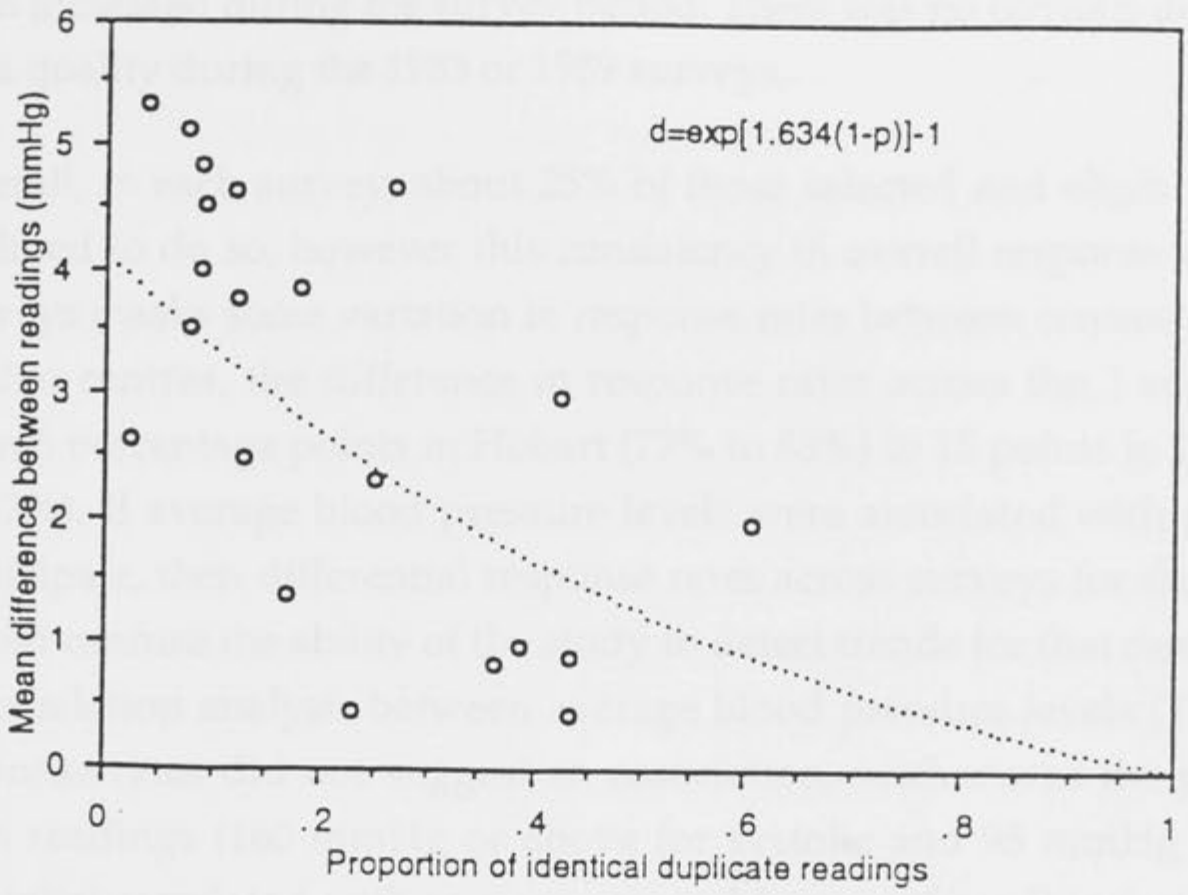

FIGURE 5.1 *Relationship between mean systolic difference (d) and proportion of identical duplicate readings (p)*

The fitted model estimates a reduction of 1.69 mmHg in the mean difference between readings as the proportion of identical readings increases from 0.1 to 0.4. This implies, for example, that a difference of 30 percentage points between centres in the proportion of identical duplicate measurements is likely to lead to a difference of 0.85 mmHg in mean systolic blood pressure, averaged over the first and second measurements (1.69/2). No such effect was apparent for diastolic measurements.

Additional analyses

Additional analyses were performed to examine whether the propensity of observers to record identical duplicate measurements had been influenced by characteristics of the respondent which may have been apparent to the observer. There was no evidence of any association between the proportion of identical duplicate measurements and the age, sex, country of birth, body mass index or socioeconomic status (as measured by level of education and occupation) of the respondent. Data to test for the effect of observer characteristics on blood pressure levels were not collected.

An analysis for trends in data quality by month of data collection suggested a deterioration in quality during the data collection period for the 1980 survey. Digit preference for zero and the proportion of identical duplicate readings

both increased during the survey period. There was no obvious deterioration in data quality during the 1983 or 1989 surveys.

Overall, in each survey, about 25% of those selected and eligible to take part declined to do so, however this consistency in overall response rates between surveys masks some variation in response rates between centres (60% to 87%). Within centres, the difference in response rates across the 3 surveys ranged from 6 percentage points in Hobart (77% to 83%) to 15 points in Brisbane (82% to 67%). If average blood pressure levels were associated with propensity to participate, then differential response rates across surveys for the same centre would confuse the ability of the study to detect trends for that centre over time. A correlation analysis between average blood pressure levels (Table 5.7) and response rates did not suggest an association, neither was the proportion of high readings (160 mmHg or above for systolic and 95 mmHg or above for diastolic) correlated with response rates. Many confounding factors are likely to have been involved however. The level of blood pressure among non-respondents is unknown.

TABLE 5.7 ***Mean blood pressure, by centre and survey***

	Systolic blood pressure			Diastolic blood pressure		
Centre	1980	1983	1989	1980	1983	1989
	mmHg					
Men						
Sydney North	135	130	130	87	76	82
Sydney South	133	140	129	83	88	83
Melbourne	136	128	133	86	78	84
Brisbane	128	134	129	87	85	84
Adelaide	139	133	130	85	86	83
Perth	135	132	128	88	83	80
Hobart	129	135	130	83	85	81
All centres	134	133	130	86	83	82
Women						
Sydney North	128	124	125	81	72	77
Sydney South	129	134	122	79	83	79
Melbourne	131	123	124	80	74	78
Brisbane	121	125	123	82	79	79
Adelaide	133	125	123	80	82	77
Perth	128	123	120	81	77	73
Hobart	123	129	125	78	81	77
All centres	127	126	123	80	78	77

Inspection of the crude mean blood pressures for men and for women in Table 5.7 reveals several inconsistencies. For example, the relatively high mean systolic value of 140 mmHg in 1983, recorded for men in Sydney South, implies an apparent increase of 7 mmHg between 1980 and 1983 followed by a decrease of 11 mmHg. The pattern is similar for women with an increase of 5

mmHg followed by a decrease of 12 mmHg. Several possible explanations for this apparently volatile behaviour have been investigated. The effect of extreme values was eliminated as the cause because median values displayed the same pattern as the means. An analysis of the Sydney South data by month of collection revealed no deterioration in calibration of the sphygmomanometer over time. An analysis by observer (701 sets of blood pressure measurements in Sydney South in 1983 were taken by one nursing sister and 166 by another) revealed patterns that are difficult to interpret (Table 5.8). After adjusting for participant's age, measurements of systolic blood pressure taken on men by Nurse 1 were 6 mmHg higher on average than measurements taken by Nurse 2 ($p<0.005$), but no such difference was apparent for measurements taken on women. For diastolic blood pressure, measurements taken on women by Nurse 1 were 4 mmHg lower than those taken by Nurse 2 ($p<0.05$).

TABLE 5.8 *Age adjusted mean blood pressure by observer, Sydney South 1983*

	Nurse 1	Nurse 2
Systolic blood pressure		
Men	141.3	135.3 #
Women	133.9	134.7
Diastolic blood pressure		
Men	87.2	89.5
Women	82.6	86.6 #
Participants		
Men	360	84
Women	341	82

# $p<0.01$

5.4 Implications

An understanding of the likely contribution of blood pressure measurement error to an analysis of trends is an important prerequisite to their interpretation. Time trends are likely to be relatively small and the sphygmomanometer, the observer and non-response bias are all sources of error which might potentially confound such an analysis.

All survey centres used standard sphygmomanometers. These were also used by the majority of centres which participated in the WHO MONICA baseline surveys (WHO MONICA Project et al. 1989). The use of random zero sphygmomanometers was considered for the 1980 survey, but it was decided that these would not be necessary provided staff involved in the survey had adequate training in the taking of blood pressures. Their use was considered again in 1983 but problems arose with several of the machines in trials before the survey, and the periodic, standardised checking of the instruments proved

impracticable. Random zero machines have been found to give lower blood pressure readings, on average, than standard mercury sphygmomanometers (De Gaudemaris et al. 1985; Dischinger and DuChene 1986; O'Brien et al. 1990; Parker et al. 1988) and higher estimates of within-person variability (Parker et al. 1988). The devices have been found to be so different that they should not be used interchangeably in epidemiological studies (De Gaudemaris et al. 1985; Parker et al. 1988). The most recent MONICA manual advocates adherence to the instrument used in earlier surveys (World Health Organization 1992). It is important, therefore, that future Australian risk factor prevalence surveys continue to use normal mercury sphygmomanometers if their estimates are to be compared longitudinally with those from existing surveys without confounding the results by type of device. If random zero machines are introduced in order to reduce observer effects, their introduction should be managed in such a way as to enable trend estimates to be adjusted for device type.

This chapter has documented *deviation from the study protocol* for blood pressure measurement, more commonly in terms of last digit preference for zero, but also in terms of the occurrence of odd digits. The simulation analysis confirms that the effect of last digit preference for zero on an analysis of trends in mean pressures is likely to be negligible. However, any analysis of trends in the prevalence rate of high blood pressure, defined as systolic and/or diastolic blood pressure above cut-off points, will need to demonstrate that there was no confounding effect of differential last digit preference for zero. The analysis makes the reasonable assumption that observers exhibited no systematic error; that is, habitual rounding in one direction, either upwards or downwards.

The analysis has also shown pronounced *variation in measurement technique* between surveys and centres. The relative frequency of identical duplicate measurements ranged from 3 to 61% (Table 5.3, systolic blood pressure). There is no gold standard of good practice to which the observed data can be compared. However, given the high biological variability of blood pressure with moment-to-moment variation, higher proportions of identical readings suggest lower precision; that is, the observer was unduly influenced by the first reading when taking the second reading, measurements were taken without due attention to accuracy, or perhaps that blood pressure was being measured once but recorded twice. The average fall in blood pressure between successive readings ranged from 0.9 to 5.3 mmHg (Table 5.6, systolic blood pressure). Although there is no evidence, it is possible that this variation could in part reflect deviation from the specified 5 minutes rest between duplicate readings (Canner et al. 1991). An association between the proportion of identical

duplicate readings and the mean difference between first and second readings is to be expected, as the two indicators are obviously not unrelated (Hense et al. 1990). The Australian data shows that absolute differences of around 30 percentage points between centres or surveys in the prevalence of identical duplicate measurement is sufficient to contribute 0.85 mmHg to trends in mean systolic blood pressure. There was no similar effect for diastolic pressure.

Both mean absolute and signed differences varied markedly between centres, and they have been advocated as important indicators of measurement quality which should be monitored periodically throughout data collection (Canner et al. 1991). Overall, the 1983 and 1989 Australian surveys showed similar recording practices and collected better quality data than the 1980 survey. The monitoring system in the latter surveys included judicious feedback on digit preference to the survey centres during the collection period and this will have helped to avoid the deterioration in data quality which occurred during the 1980 survey. Inspection of mean blood pressure levels (Table 5.7) revealed trends which are difficult to interpret. The treatment of the data for Sydney South and other cities with equally implausible changes in population means is problematic for any analysis of trends in blood pressure. They could reflect a systematic bias by an observer or a faulty sphygmomanometer but these effects are difficult to substantiate.

In conclusion, this chapter has demonstrated that epidemiological studies need to be mindful of the effects of non-sampling errors on comparisons of blood pressure distributions. The effect of last digit preference for zero, more common in the 1980 survey, is likely to be negligible on estimates of mean and standard deviation but could contribute substantially to estimates above cut-off points. The tendency to record identical duplicate measurements has the potential to confound differences in mean levels of systolic blood pressure between populations. Estimates suggest that this tendency might contribute up to 0.85 mmHg to observed differences in systolic blood pressure between some centres and could represent a significant contribution to estimates of time trends and geographic differences in systolic blood pressure levels.

Chapter 4 reported overall changes in systolic blood pressure of -4.1 mmHg in men and -3.8 mmHg in women (with 95% confidence intervals of -4.9, -3.2 mmHg and -4.6, -3.0 mmHg respectively). The difference of 20 percentage points between 1989 (37%) and 1980 (17%) in the proportion of identical duplicate measurements (Table 5.3) is likely to have contributed 0.5 mmHg[1] to

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[1] Calculated from Figure 5.1 as [d(0.17)-d(0.37)]/2 = [2.88-1.80]/2 = 0.54

these estimates, that is approximately 12%. It is concluded that the trends observed represent real decreases in the average level of systolic blood pressure among Australian adults.

Generally, deviation from the measurement protocol and variation in measurement technique are likely to impair the ability of the study to properly detect trends and should be minimised. To this end, the training procedures for blood pressure measurement which have been followed in past risk factor prevalence surveys should be reviewed before any future such surveys are conducted. In addition, the indicators used in this analysis should be monitored closely during the course of the data collection. Reassuringly, there is no evidence that observers were influenced in their recording practice by the respondents' characteristics.

Chapter Six

Inequalities in risk factors and cardiovascular mortality among Australia's immigrants

6.1 Introduction

The Australian population embraces a wide range of origins and cultures. Over 20% of residents were born overseas, representing over 100 different countries of birth (Australian Bureau of Statistics 1987). Inspection of recent settler arrival statistics (Bureau of Immigration Research 1995) shows that the European sources of post-war immigration such as Italy, Yugoslavia and Greece have been replaced by settlers from Asian countries, notably Hong Kong and Vietnam (Table 6.1). British immigrants now represent less than 20% of recent arrivals compared with over 50% in past years.

TABLE 6.1 *Top ten immigrant groups in 1986 (a), and settler arrivals in 1990-91 (b)*

Population 1986		Settler arrivals 1990-91	
Birthplace	Per cent	Birthplace	Percent
United Kingdom and Ireland	32.3	United Kingdom and Ireland	18.0
Italy	7.5	Hong Kong	11.1
New Zealand	6.1	Vietnam	10.9
Yugoslavia	4.3	New Zealand	6.1
Greece	3.9	Philippines	5.3
Germany	3.3	Malaysia	4.7
Netherlands	2.7	India	4.2
Vietnam	2.4	Taiwan	2.9
Poland	1.9	Sri Lanka	2.7
Lebanon	1.6	China	2.7
Other	34.0	Other	31.5
Total	100.0	Total	100.0

(a) 1986 was a census year which fell within the period of the risk factor data. (b) Data for 1990-91 are illustrative of recent arrival distributions.

Many studies of the health of immigrants in Australia have focused on mortality data, often for selected causes of death or specific immigrant groups. One exception is the analysis of 1980-82 mortality data by Young which covered a relatively wide range of causes and immigrant groups (Young 1986). The analysis showed that immigrants to Australia have generally experienced lower mortality rates than people born in Australia across a wide range of causes, including cardiovascular disease. A more recent analysis of cardiovascular mortality data for 1987-89 showed a lower age standardised mortality ratio (SMR) for immigrants from Vietnam,

Greece, Italy, Malaysia, China, Philippines (women) and Central and South America (Table 6.2). High SMRs were found for immigrants from Malta (women only), Poland and the Pacific Islands (Young 1992b).

TABLE 6.2 ***SMRs for cardiovascular disease by birthplace group, 1987-89***

Birthplace group	Men	Women
United Kingdom and Ireland	92*	92*
Greece	62*	55*
Italy	67*	68*
Yugoslavia	87*	79*
Malta	103	124*
Germany	95	90
Netherlands	91	84*
Poland	124*	117*
Lebanon	80*	118
Egypt	90	122
Vietnam	30*	35*
Malaysia	71*	59*
Philippines	81	56*
India	92	80
China	62*	61*
Hong Kong and Macao	33*	
South Africa	89	85
Canada	96	66
United States of America	96	109
Central and South America	54*	69*
New Zealand	109	105
Other Oceania	137*	165*
Australia	103*	104*

* SMR significantly different from 100 ($p<.05$)
Source: Young 1992b

The extent to which mortality and morbidity (Australian Bureau of Statistics 1991; Young 1992a) differentials have been influenced by differences in cardiovascular risk factor levels is not clear, as there have been few comprehensive studies of risk factor differentials among Australia's immigrant groups. Differentials are to be expected. Recent results from the WHO MONICA Project demonstrated marked variation in cardiovascular risk factor levels between population centres in 26 countries (WHO MONICA Project et al. 1989), many of which are significant sources of Australia's immigrants.

This chapter systematically compares the levels of biomedical and behavioural risk factors among a range of immigrant groups in Australia, using native-born Australians as a reference group. Variation in risk factor levels by period of residence is examined. Also, risk factor profiles for each immigrant group are compared with their cardiovascular mortality experience. The Risk Factor Prevalence Study provided relatively recent cardiovascular risk factor data on over 6,000 immigrants.

6.2 Methods

Definition of immigrant groups and period of residence

Each survey included the question `Where were you born?' and respondents were asked to write State or Territory if born in Australia, or country if born overseas. Responses to the 1989 survey were coded to the Australian Standard Classification of Countries for Social Statistics which was released in 1990 (Australian Bureau of Statistics 1990). The vast majority of responses to the 1980 and 1983 surveys were able to be successfully recoded to this classification. Aggregation of birthplaces into immigrant groups was necessary in order to provide reasonable sample sizes for the analysis (Table 6.3). Countries which have historically been a major source of migrants to Australia have been separately identified. Immigrants from Scotland and Ireland, who have been identified as having higher overall mortality than Australian-born (Young 1986), were analysed separately from the rest of the United Kingdom. It was possible to separate Asia, which covers such a diverse group of nations, into two broad groups; Southeast Asia and Other Asia. Sample numbers did not the separate identification of Northeast Asia and South Asia.

TABLE 6.3 *Sample counts by immigrant group, for the three surveys combined*

Immigrant group	Men	Women
England and Wales	970	879
Scotland and Ireland	260	213
Greece	194	162
Italy	321	278
Other Southern Europe (a)	202	208
Western Europe (b)	289	260
Eastern Europe (c)	263	206
Middle East and Nth Africa (d)	131	92
Southeast Asia (e)	146	171
Other Asia (f)	137	136
New Zealand	143	138
Other countries (g)	121	121
Insufficient information (h)	37	38
Australian-born	7137	7804
Total	*10351*	*10706*

(a) Includes Yugoslavia, Cyprus and Malta (b) Includes Germany, Netherlands and Austria (c) Northern Europe, Eastern Europe, the USSR and the Baltic States. Includes Poland, Hungary and Finland (d) Includes Lebanon, Egypt and Turkey (e) Includes Vietnam, Indonesia, Malaysia and the Philippines (f) Includes India, China, Taiwan and Hong Kong (g) Includes South Africa, Canada, USA and PNG (h) United Kingdom, Great Britain, etc.

In addition to reporting country of birth, respondents born overseas were also asked how many years they had lived in Australia. To allow for the different waves of immigrants to Australia, the pattern of risk factor levels by period of residence was estimated separately for four broad regions. The

classification into regions and period of residence was the best possible given the relatively few recent immigrants from Europe in the sample, and few longer term Asian residents.

Statistical analysis

The purpose of the analysis was to detect differences in risk factor levels between each immigrant group and Australian-born men and women. The null hypotheses were that no such differences existed.

For continuous variables (e.g. blood pressure), inequalities between immigrant groups were examined by analysis of covariance with age as a continuous covariate. Survey year, location and their interaction were also included in the model to allow for the study design. Consideration was given to including an age by immigrant group interaction term in the model to allow for different slopes in the relationship between age and the risk factor for each immigrant group. The term was intermittently and marginally significant across risk factor by sex combinations and it was decided that the sample size could not sustain the added complexity to the model. Interpretation was also unclear, given the close association between age and years since immigration.

The analysis shows, for each immigrant group, the crude (unadjusted) mean and the adjusted differential (D1), which is the immigrant mean minus the Australian-born mean adjusted for age and study design factors. Statistically significant differences (2-sided tests) have been indicated. Body mass index, smoking status, alcohol consumption and oral contraceptive use (women) were treated as potential explanatory variables, to explore possible reasons for the differentials (D1). The differentials which resulted from the addition of these covariates to the model have been denoted by D2.

Multiple logistic regression was used to analyse categorical variables (eg. current smoking). The model included all survey design factors and treated age as a continuous covariate. The parameter estimates from the model were used to derive prevalence odds ratios for each immigrant group, and their 95% confidence limits, based on the Australian-born as the reference group.

6.3 Differentials between immigrant groups

Results for diastolic blood pressure and triglyceride are not given in tabular form but statistically significant differentials are mentioned in the text.

Blood pressure

After adjusting for age and study design factors, mean systolic blood pressure was lower in male immigrants from the Middle East (-5.4 mmHg), Other Asia (-5.3 mmHg), Southeast Asia (-4.4 mmHg), Greece (-3.6 mmHg), Italy (-1.9 mmHg) and England and Wales (-1.9 mmHg) than their Australian-born counterparts (Table 6.4). Only men from Eastern Europe had statistically higher systolic blood pressure (+2.4 mmHg) than Australian-born men. Differentials for diastolic blood pressure in men were largely non-existent.

TABLE 6.4 ***Systolic blood pressure and body mass index by immigrant group***

	Systolic blood pressure				Body mass index (a)			
Immigrant group	Mean	D1	SE(D1)	D2	Mean	D1	SE(D1)	D2
	mmHg				kg/m^2			
Men								
England and Wales	131.1	-1.9 #	0.6	-0.9	24.9	-0.6 #	0.1	-0.6 #
Scotland and Ireland	132.1	-1.5	1.0	-0.8	25.0	-0.6	0.2	0.6 *
Greece	130.8	-3.6 #	1.2	-4.7 #	27.1	1.5 #	0.3	1.6 #
Italy	133.8	-1.9*	0.9	-2.9 #	26.8	1.2 #	0.2	1.3 #
Other Southern Europe	134.4	0.3	1.1	-0.2	26.3	0.8 #	0.3	0.9 #
Western Europe	132.3	-1.3	1.0	-1.2	25.8	0.1	0.2	0.1
Eastern Europe	140.7	2.4*	1.0	1.4	26.6	0.8 #	0.2	0.8 #
The Middle East	127.9	-5.4 #	1.4	-5.3 #	26.2	0.9 #	0.3	1.0 #
Southeast Asia	124.3	-4.4 #	1.3	-1.6	23.4	-1.8 #	0.3	-1.7 #
Other Asia	127.4	-5.3 #	1.4	-2.7	23.9	-1.6 #	0.3	-1.5 #
New Zealand	127.6	-2.5	1.4	-2.2	25.2	-0.1	0.3	-0.1
Other countries	129.1	-1.8	1.5	-0.4	24.8	-0.6	0.3	-0.6
Australian-born	*131.7*				*25.4*			
Women								
England and Wales	123.9	-2.3 #	0.6	-1.7 #	23.8	-0.5 #	0.2	-0.5 #
Scotland and Ireland	127.8	-0.7	1.1	0.1	24.1	-0.4	0.3	-0.3
Greece	124.5	-1.9	1.2	-4.5 #	26.9	2.7 #	0.4	2.2 #
Italy	127.7	-0.3	1.0	-1.9	26.6	2.3 #	0.3	2.1 #
Other Southern Europe	127.7	0.8	1.1	-1.2	26.6	2.3 #	0.3	2.1 #
Western Europe	126.7	-0.4	1.0	0.1	24.7	0.3	0.3	0.3
Eastern Europe	133.3	1.8	1.1	2.1	25.3	0.6	0.3	0.5
The Middle East	120.3	-4.3 #	1.6	-6.3 #	26.2	2.5 #	0.5	2.2 #
Southeast Asia	116.1	-3.4 #	1.2	-2.2	22.3	-1.3 #	0.4	-1.7 #
Other Asia	122.1	-2.2	1.4	-1.2	23.0	-0.9 *	0.4	-1.1 #
New Zealand	119.6	-1.6	1.3	-1.6	23.3	-0.3	0.4	-0.1
Other countries	121.8	-1.8	1.4	-2.2	24.3	0.4	0.4	0.3
Australian-born	*124.6*				*24.1*			

(a) Excludes pregnant women — Mean Crude (unadjusted) mean

D1 Immigrant group mean minus the Australian-born mean, adjusted for age and study design factors

SE(D1) Standard error of D1.

D2 D1 further adjusted for covariates (body mass index, smoking status, alcohol consumption and oral contraceptive use as appropriate).

* $p<0.05$, # $p<0.01$ where p is the probability of a difference of this magnitude or greater if the null hypothesis was true.

Systolic blood pressure was lower among women from the Middle East (-4.3 mmHg), Southeast Asia (-3.4 mmHg) and England and Wales (-2.3 mmHg) compared with Australian-born women As for men, the differentials for diastolic blood pressure were noticeably less pronounced than those for

systolic blood pressure, and only the differences for those women from the Middle East (-2.1 mmHg) and England and Wales (-1.5 mmHg) reached statistical significance. Although there was some variation between immigrant groups in the proportion on treatment for blood pressure, the differentials which have been identified were also apparent when the analysis was confined to those not on treatment.

Overall obesity

The results for body mass index suggest marked differences in overall obesity between immigrant groups. Relative to native-born Australians, men from the Asian regions and from the United Kingdom had a statistically significantly lower mean body mass index (Table 6.4). Results were similar but less pronounced for women. Men and (particularly) women from Southern European regions and the Middle East each had higher adjusted body mass index on average than their native-born counterparts, as did men from Eastern Europe.

Blood lipids

TABLE 6.5 ***Total plasma cholesterol and HDL cholesterol by immigrant group***

	Total cholesterol (a)				HDL cholesterol (a)			
Immigrant group	Mean	D1	SE(D1)	D2	Mean	D1	SE(D1)	D2
		mmol/L				mmol/L		
Men								
England and Wales	5.67	-0.02	0.04	0.01	1.23	0.00	0.01	-0.00
Scotland and Ireland	5.75	0.04	0.07	0.03	1.24	0.01	0.02	-0.00
Greece	5.63	-0.08	0.08	-0.13	1.15	-0.07 #	0.02	-0.01
Italy	5.57	-0.19 #	0.07	-0.23 #	1.21	-0.02	0.02	0.03
Other Southern Europe	5.54	-0.14	0.08	-0.16	1.18	-0.04	0.03	-0.01
Western Europe	5.91	0.17 *	0.07	0.16 *	1.19	-0.04	0.02	-0.02
Eastern Europe	5.87	-0.02	0.07	-0.06	1.25	0.02	0.02	0.04
The Middle East	5.67	0.05	0.10	0.01	1.09	-0.13 #	0.03	-0.06 *
Southeast Asia	5.45	-0.07	0.09	0.06	1.18	-0.03	0.03	-0.04
Other Asia	5.68	0.01	0.10	0.12	1.18	-0.03	0.03	-0.04
New Zealand	5.50	-0.08	0.09	-0.07	1.26	0.04	0.03	0.04
Other countries	5.63	-0.00	0.10	0.05	1.19	-0.03	0.03	-0.03
Australian-born	*5.66*				*1.23*			
Women								
England and Wales	5.65	0.00	0.04	0.02	1.52	-0.00	0.01	-0.01
Scotland and Ireland	5.72	-0.06	0.07	-0.07	1.49	-0.05	0.03	-0.04
Greece	5.48	-0.10	0.09	-0.09	1.34	-0.16 #	0.03	-0.08 *
Italy	5.44	-0.25 #	0.07	-0.25 #	1.43	-0.10 #	0.02	-0.04
Other Southern Europe	5.61	-0.02	0.08	-0.01	1.43	-0.09 #	0.03	-0.04
Western Europe	5.73	0.05	0.07	0.01	1.52	-0.01	0.02	0.00
Eastern Europe	5.90	0.00	0.08	0.03	1.51	-0.02	0.03	0.02
The Middle East	5.48	-0.02	0.11	-0.05	1.33	-0.19 #	0.04	-0.10 *
Southeast Asia	5.16	-0.14	0.09	-0.06	1.45	-0.03	0.03	-0.05
Other Asia	5.55	0.03	0.09	0.11	1.43	-0.08 *	0.03	-0.07 *
New Zealand	5.30	-0.08	0.09	-0.11	1.57	0.06	0.03	0.03
Other countries	5.31	-0.14	0.10	-0.16	1.47	-0.03	0.04	-0.03
Australian-born	*5.58*				*1.52*			

(a) Fasting; See also notes to Table 6.3

Only men and women from Italy and men from Western Europe had adjusted mean levels of total cholesterol which were significantly different from their Australian-born counterparts (-0.19, -0.25 and +0.17 mmol/L respectively, Table 6.5). Statistically significantly lower levels of HDL cholesterol were observed among men and women from Greece and the Middle East, and among women from Italy, Other Southern Europe, and Other Asia.

Men and women from Italy had lower levels of LDL cholesterol than their Australian-born counterparts (Table 6.6). Only men from Western Europe had a higher adjusted mean level of LDL cholesterol (+0.20 mmol/L).

TABLE 6.6 ***LDL cholesterol and TC/HDL ratio by immigrant group***

	LDL cholesterol				TC/HDL ratio (a)			
Immigrant group	Mean	D1	SE(D1)	D2	Mean	D1	SE(D1)	D2
	mmol/L							
Men								
England and Wales	3.83	0.01	0.03	0.02	4.88	-0.05	0.06	0.00
Scotland and Ireland	3.85	0.02	0.06	0.03	4.93	-0.02	0.10	0.02
Greece	3.87	0.04	0.07	-0.03	5.17	0.19	0.12	-0.13
Italy	3.75	-0.12*	0.06	-0.17#	4.92	-0.08	0.10	-0.32#
Other Southern Europe	3.67	-0.12	0.08	-0.15	4.97	0.04	0.12	-0.14
Western Europe	4.03	0.18#	0.06	0.15*	5.24	0.28#	0.10	0.21*
Eastern Europe	3.92	-0.03	0.06	-0.09	4.99	-0.09	0.11	-0.21*
The Middle East	3.89	0.13	0.09	0.05	5.45	0.57#	0.14	0.21
Southeast Asia	3.64	-0.04	0.08	0.02	4.89	0.03	0.14	0.16
Other Asia	3.84	0.04	0.09	0.09	5.07	0.13	0.14	0.23
New Zealand	3.61	-0.11	0.08	-0.11	4.65	-0.21	0.14	-0.19
Other countries	3.82	0.06	0.09	0.09	5.17	0.30	0.15	0.36*
Australian-born	*3.78*				*4.89*			
Women								
England and Wales	3.65	0.00	0.04	0.02	3.95	0.03	0.05	0.07
Scotland and Ireland	3.71	-0.03	0.07	-0.05	4.09	0.11	0.09	0.07
Greece	3.67	0.07	0.08	0.02	4.30	0.36#	0.11	0.16
Italy	3.53	-0.14*	0.06	-0.18#	4.01	0.07	0.09	-0.09
Other Southern Europe	3.64	0.04	0.07	0.01	4.09	0.18	0.10	0.04
Western Europe	3.71	0.05	0.06	0.02	4.02	0.09	0.09	0.04
Eastern Europe	3.86	0.02	0.07	0.04	4.12	0.06	0.10	-0.06
The Middle East	3.58	0.06	0.11	-0.01	4.34	0.51#	0.15	0.27
Southeast Asia	3.21	-0.16	0.08	-0.07	3.71	-0.10	0.11	0.02
Other Asia	3.66	0.10	0.09	0.15	4.09	0.23*	0.12	0.28*
New Zealand	3.32	-0.11	0.08	-0.12	3.52	-0.23	0.12	-0.18
Other countries	3.40	-0.09	0.09	-0.11	3.80	-0.04	0.12	-0.06
Australian-born	*3.60*				*3.88*			

(a) Fasting; See also notes to Table 6.3

Men and women from New Zealand had more beneficial TC/HDL ratios than their Australian-born counterparts but the differences did not reach statistical significance. Men from Western Europe and the Middle East, and women from Greece, Other Southern Europe, the Middle East and Other

Asia all had statistically significantly higher ratios than Australian-born men and women.

The mean triglyceride level (not shown) among women from the Middle East was highly statistically significantly different (+0.26 mmol/L, $p<.01$) from their Australian-born counterparts. Other significant triglyceride differentials ($p<.05$) were men and women from Italy (-0.14 and +0.09 mmol/L), and women from Other Southern Europe (+0.10 mmol/L) and Southeast Asia (+0.11 mmol/L).

Smoking status

The prevalence of current smoking of any form of tobacco was significantly higher among men and women from Scotland and Ireland (adjusted prevalence odds ratios 1.65 and 1.63 respectively, Table 6.7). Smoking prevalence odds ratios were also significantly higher among men from Southern Europe (Greece 1.65; Italy 1.60; and Other Southern Europe 1.80) and the Middle East (2.03), and women from Western Europe (1.39). Women from Greece (0.41), Italy (0.43), Southeast Asia (0.33) and Other Asia (0.66) had lower smoking prevalence odds ratios than Australian-born women.

Alcohol consumption

Light alcohol consumption (1-27 drinks per week), potentially a beneficial risk factor, was reported more commonly among male immigrants from England and Wales, Italy, Western Europe and Eastern Europe (adjusted prevalence odds ratios of 1.58, 1.60, 1.96 and 1.63 respectively). Prevalence odds ratios were significantly lower among male immigrants from the Middle East (0.51) and Southeast Asia (0.64).

Among women, the prevalence odds ratios for light drinking (1-13 drinks per week) were higher among immigrants from England and Wales (1.34), and lower among immigrants from Southern European countries (0.25, 0.52, 0.50), the Middle East (0.29) and Asian countries (0.25 and 0.38).

Exercise during leisure-time

Physical inactivity during leisure-time was more prevalent among men and women from Southern European counties, Eastern European countries and the Middle East than their Australian-born counterparts (Table 6.7). Exercise was also relatively unpopular among men and women from Southeast Asia and women from Other Asia, however men from the latter Asian countries

exercised as frequently as Australian-born men. Men from Scotland and Ireland also reported a higher prevalence odds ratio for physical inactivity.

TABLE 6.7 ***Behavioural risk factors (a) by immigrant group***

	Smoking			Light alcohol			Physical inactivity		
Immigrant group	%	OR	95% CI	%	OR	95% CI	%	OR	95% CI
Men									
England and Wales	31.4	1.03	0.88-1.19	84.2	1.58#	1.32-1.90	27.0	0.99	0.84-1.16
Scotland and Ireland	42.5	1.65#	1.28-2.13	78.1	1.10	0.88-1.48	36.0	1.51#	1.12-2.04
Greece	41.8	1.65#	1.23-2.22	80.4	1.33	0.92-1.92	55.7	3.35#	2.39-4.68
Italy	41.9	1.60#	1.27-2.02	82.5	1.60#	1.19-2.16	61.2	3.92#	2.95-5.20
Other Southern Europe	44.3	1.80#	1.35-2.40	80.7	1.32	0.92-1.90	58.3	3.67#	2.62-5.13
Western Europe	34.9	1.21	0.94-1.55	86.5	1.96#	1.38-2.76	33.2	1.31	0.97-1.75
Eastern Europe	32.4	1.11	0.85-1.46	81.4	1.63#	1.18-2.25	39.2	1.55#	1.13-2.14
The Middle East	46.6	2.03#	1.43-2.89	64.1	0.51#	0.35-0.74	48.5	2.67#	1.78-4.01
Southeast Asia	33.6	1.09	0.76-1.55	70.6	0.64*	0.44-0.92	46.0	2.77#	1.92-3.99
Other Asia	35.8	1.27	0.89-1.82	77.4	0.98	0.65-1.47	27.3	1.06	0.67-1.68
New Zealand	24.5	0.69	0.47-1.02	84.6	1.57	0.99-2.50	22.2	0.83	0.52-1.32
Other countries	28.1	0.90	0.60-1.35	82.6	1.30	0.81-2.11	30.4	1.28	0.83-1.97
Australian-born	*31.4*	*1.00*		*77.7*	*1.00*		*26.3*	*1.00*	
Women									
England and Wales	24.2	1.03	0.87-1.21	77.2	1.34#	1.13-1.59	26.6	0.89	0.74-1.07
Scotland and Ireland	33.3	1.63#	1.21-2.18	76.1	1.35	0.98-1.86	22.7	0.68	0.46-1.00
Greece	11.8	0.41#	0.25-0.66	40.7	0.25#	0.18-0.35	57.6	3.45#	2.37-5.03
Italy	11.9	0.43#	0.30-0.62	56.8	0.52#	0.40-0.66	57.2	3.29#	2.46-4.40
Other Southern Europe	18.7	0.72	0.50-1.02	56.7	0.50#	0.38-0.67	65.2	4.78#	3.41-6.70
Western Europe	29.6	1.39*	1.06-1.83	75.8	1.27	0.96-1.70	26.8	0.89	0.64-1.24
Eastern Europe	23.8	1.04	0.75-1.45	66.3	0.85	0.63-1.15	40.3	1.56*	1.10-2.22
The Middle East	29.4	1.21	0.76-1.91	47.8	0.29#	0.19-0.44	46.9	2.62#	1.58-4.35
Southeast Asia	9.9	0.33#	0.20-0.55	42.7	0.25#	0.18-0.34	56.0	3.71#	2.62-5.26
Other Asia	16.2	0.66*	0.38-0.95	52.9	0.38#	0.27-0.54	51.6	3.00#	2.00-4.52
New Zealand	26.8	1.13	0.77-1.66	81.2	1.43	0.92-2.21	18.7	0.67	0.41-1.09
Other countries	18.2	0.68	0.43-1.09	68.6	0.78	0.53-1.16	35.1	1.49	0.97-2.30
Australian-born	*24.4*	*1.00*		*73.2*	*1.00*		*27.5*	*1.00*	

(a) Current smoking, light alcohol intake and physical inactivity. Light alcohol intake is 1-27 drinks/week for men, 1-13 drinks/week for women. Physical inactivity is no leisure-time activity of any kind during past 2 weeks.

% Crude (unadjusted) percentages.

OR Prevalence odds ratio adjusted for age and study design factors.

* $p<0.05$, # $p<0.01$, where p is the probability of an odds ratio of this magnitude or greater if the null hypothesis was true.

Adjustment for covariates

The extent to which body mass index, smoking status, alcohol consumption, and use of oral contraception (women) modified the differentials for blood pressure and blood lipids can be seen by comparing D1 and D2 (Tables 6.4-6.6). Adjustment for the covariates sometimes amplified and sometimes `explained' the differentials. Higher body mass index was associated with greater blood pressure, as was heavy alcohol consumption. The association between smoking status and blood pressure was stronger in women than men, with current smokers having lower systolic and diastolic pressures. Body mass index also exhibited significant positive associations with total cholesterol, triglyceride and LDL cholesterol, and significant negative

associations with HDL cholesterol and TC/HDL. Other significant relationships were between alcohol consumption and HDL cholesterol (heavy intake associated with higher levels) and TC/HDL (heavy intake associated with lower levels); and between smoking status and HDL cholesterol (current smokers had lower levels) and TC/HDL (current smokers had higher levels). For women, taking oral contraceptives was associated with statistically significantly higher total cholesterol, lower HDL cholesterol, higher triglyceride, higher LDL cholesterol and higher TC/HDL.

Period of residence

The difference in systolic blood pressure between shorter and longer stay male immigrants from the UK and Ireland (+2.1 mmHg) was statistically significant ($p<.01$) as was the increase in systolic blood pressure for (other) Europeans (+3.3 mmHg) (Table 6.8). Among women, the more recently arrived immigrants from the UK and Ireland and from Asia had significantly lower systolic blood pressure than Australian-born women, but not so the longer stay immigrants from these regions. None of the comparisons by period of residence showed statistically significant trends for women, except the trend for `all immigrants' (from -2.8 mmHg to -0.9 mmHg). None of the comparisons for association between period of residence and diastolic blood pressure were statistically significant.

Several differentials for body mass index were statistically significant; however, period of residence was associated with statistically significant increases only for men and women from Asia (+0.9 kg/m^2, $p<.01$; +1.0 kg/m^2, $p<.05$ respectively). Differentials for total cholesterol were small and varied little by period of residence in Australia. Similarly, there was no evidence of change in HDL cholesterol, triglyceride or LDL cholesterol levels with period of residence. The trend in TC/HDL for European males was statistically significant ($p<.01$).

None of the tests for association between smoking prevalence and period of residence were statistically significant. For light alcohol intake, longer period of residence was associated with a statistically significant fall in prevalence for men from the UK and Ireland and an increase in prevalence for men and women from Asia and women from Europe.

Physical inactivity during leisure-time was more common among immigrants from Europe and Asia than their Australian-born counterparts. The decreases in prevalence odds ratios between immigrants with shorter and longer residential periods were statistically significant for men and

women from Asia, and women from Europe. Standardising across region of birth showed that exercise was more popular among longer stay immigrants than shorter stay immigrants.

TABLE 6.8 *Risk factor differentials (a) by period of residence and immigrant group*

Period of residence	n	SBP	BMI	TC	HDL	LDL	TC/HDL	SMK	LTA	INA
		mmHg	kg/m^2	mmol/L	mmol/L	mmol/L		— odds ratio (b) —		
Men										
UK and Ireland										
≤15 years	395	-3.3#	-0.7#	+0.07	+0.01	+0.08	+0.01	1.03	1.89#	1.04
≥16 years	867	-1.2*	-0.6#	-0.05	+0.00	-0.01	-0.07	1.20#	1.31#	1.10
Europe (c)										
≤15 years	132	-3.7#	+0.3	-0.06	+0.02	-0.07	-0.10	1.20	2.55#	2.29#
≥16 years	1127	-0.4	+0.9#	-0.04	-0.03#	+0.00	+0.08	1.45#	1.51#	2.43#
Asia (d)										
≤15 years	237	-5.1#	-1.3#	-0.01	-0.06#	+0.02	+0.24*	1.61#	0.56#	3.07#
≥16 years	176	-4.8#	-0.4	+0.01	-0.07#	+0.07	+0.22	1.12	0.88	1.11
Other (e)										
≤15 years	139	-1.9	-0.1	-0.04	-0.02	+0.00	+0.19	0.91	1.23	1.19
≥16 years	117	-2.7	-0.6*	+0.01	+0.04	-0.03	-0.12	0.62*	1.67	0.79
All immigrants										
≤15 years	903	-3.4#	-0.5#	-0.01	-0.01	+0.01	+0.09	1.16	1.35#	1.72#
≥16 years	2287	-2.3#	-0.2	-0.02	-0.01	+0.01	+0.03	1.04	1.30#	1.24*
Women										
UK and Ireland										
≤15 years	359	-2.9#	-0.6*	-0.08	-0.00	-0.07	-0.04	1.16	1.40*	0.92
≥16 years	764	-1.2	-0.4*	+0.04	-0.03	+0.04	+0.13*	1.11	1.33#	0.82*
Europe (c)										
≤15 years	161	-1.8	+1.4#	-0.09	-0.10#	+0.02	+0.21	1.03	0.44#	3.96#
≥16 years	949	+0.3	+1.6#	-0.06	-0.06#	-0.01	+0.12#	0.74#	0.65#	2.14#
Asia (d)										
≤15 years	242	-4.1#	-0.7*	-0.11	-0.10#	-0.07	+0.15	0.51#	0.21#	4.74#
≥16 years	156	-1.8	+0.3	+0.04	-0.06*	+0.07	+0.17	0.74	0.49#	1.74#
Other (e)										
≤15 years	139	-2.4	-0.1	-0.15	+0.02	-0.15	-0.15	0.89	0.91	1.22
≥16 years	111	-1.0	+0.1	-0.04	+0.03	-0.04	-0.14	0.90	1.21	0.86
All immigrants										
≤15 years	901	-2.8#	+0.0	-0.11	-0.05#	-0.07	0.04	0.86	0.59#	2.14#
≥16 years	1980	-0.9	+0.4*	-0.01	-0.03*	+0.02	0.07	0.86	0.84*	1.27#

(a) The difference between each immigrant group estimate and the Australian-born estimate, adjusted for age and study design factors. Differentials for `all immigrants' are adjusted for immigrant group composition.
(b) Prevalence odds ratios for current smoking (SMK), light alcohol intake (LTA) and physical inactivity (INA)
(c) Excludes UK and Ireland. (d) Includes the Middle East and North Africa.
(e) Includes New Zealand. * $p<0.05$, # $p<0.01$

6.4 Immigrant group profiles

Although it is generally accepted that cardiovascular disease is related substantially to the risk factors considered in this analysis, the relationship is exceedingly complex and is made more so by the additional factors associated with immigration. The differentials which have been identified are likely to be a function of the risk factor profile of the source populations, the selection effect in the migration process, environmental influences after immigration and the degree to which habits and customs of the previous country have been maintained. Nevertheless, it is instructive to explore the extent to which differentials in biomedical and life-style risk factors among immigrant groups correlate with cardiovascular mortality experience.

The British and Irish

The British and Irish immigrants are not a homogeneous group. Immigrants from Scotland and Ireland reported higher prevalence rates of smoking and physical inactivity (men), those from England and Wales had lower blood pressure levels, and higher prevalence rates of light alcohol intake compared with Australian-born. These results are consistent with previous immigrant mortality analyses which have shown higher total mortality (Young 1986) and cardiovascular mortality (Stenhouse and McCall 1970) among the Scots and Irish than the English and Welsh. Overall, the British and Irish also had lower body mass index than Australian-born men and women and their risk factor profile was consistent with lower SMRs from cardiovascular disease (Table 6.1).

Southern Europeans

Greek and Italian male immigrants had lower systolic blood pressure than Australian-born men despite significantly higher body mass index. There was no evidence of lower systolic blood pressure among women from Southern European countries. Previous Australian studies have found lower levels among Italian immigrants (Ulman and Abernethy 1975; Armstrong et al. 1983). Southern European immigrants in general (English and Bennett 1985), and Italian immigrants in particular (Armstrong et al. 1983), are known to have higher mean body mass index than other immigrants or Australian-born men and women. This present analysis demonstrated the characteristic independently for immigrants from Greece, Italy and Other Southern Europe. The significantly lower levels of total cholesterol among Italian immigrants compared with Australian-born men and women is a new finding (Armstrong et al. 1983 found no difference). Women from each Mediterranean region had lower HDL cholesterol levels than Australian-born women, consistent with their alcohol drinking behaviour but not their smoking habits.

Regarding life-style factors, each Mediterranean region reported a significantly higher prevalence rate of leisure-time inactivity compared with Australian-born men and women. The marked sex differential in smoking prevalence rates has been noted previously among Italian immigrants (Armstrong et al. 1983). Women from each region reported significantly lower prevalence rates of light alcohol intake than Australian-born women. The men tended to have higher rates than Australian-born men although statistical significance was reached only for men from Italy.

Cardiovascular mortality rates have been persistently lower among immigrants from Southern European countries compared with Australian men and women and remain so (Table 6.2). For immigrants from Greece, the risk factor profile of men suggests that factors other than the traditional risk factors considered in this analysis are important. Greek men have advantageously lower systolic blood pressure, but lower HDL cholesterol, higher body mass index, higher smoking prevalence rate and higher leisure time inactivity. Greek women have a lower prevalence of smoking, but all other significant risk factor differentials are disadvantageous. Results for immigrants from Italy correlate better with traditional wisdom. The men have advantageous differentials for systolic blood pressure, total cholesterol, LDL cholesterol and light alcohol intake. The women have advantageous total and LDL cholesterol levels, and lower smoking prevalence.

The lipid results are not entirely consistent with the hypothesis that the Mediterranean diet offers a protective mechanism against cardiovascular disease. The low mortality rates for those from the Mediterranean region coincides with a diet which is relatively high in fresh fruit and vegetables, and high in olive oil. The olive oil used in the diets of people in Italy and Greece is around 80% oleic acid (Keys et al. 1986) and substitution of dietary saturated fatty acids with oleic acid results in a fall in LDL cholesterol levels (Carleton et al. 1991). Only amongst Italian immigrants was there evidence of lower LDL cholesterol levels among Southern European immigrants in Australia. Overall, the blood lipid profile of immigrants from Southern European countries was not markedly different from that of men and women born in Australia.

Light and moderate alcohol consumption has been linked to a reduced risk of coronary heart disease (Marmot and Brunner 1991; Jackson et al. 1991). In the present analysis, men from Southern Europe generally had a higher prevalence rate of light alcohol intake than men born in Australia but the converse was true for Southern European women compared with Australian-born women (Table 6.7). Various mechanisms have been proposed for the protective effect of moderate alcohol consumption. Alcohol intake is known to raise HDL cholesterol levels but neither men from Italy, Greece or other Southern European countries had higher HDL cholesterol levels than Australian-born men. Other possible mechanisms are that moderate alcohol consumption inhibits blood coagulation through reduced plasma fibrinogen concentrations (Meade et al. 1979), beneficial effects on the fibrinolytic system (Hendricks et al. 1994) and reduced platelet

reactivity (Renaud and De Lorgeril 1992). Some studies have found that type of alcoholic beverage is more important (Gronbaek et al. 1995), in particular total wine ethanol intake (Criqui and Ringel 1994). Wine plays an important part in the main meals of Italians and Greeks and some researchers have speculated that it is the consumption of wine in modest amounts with meals, and the slower absorption, which has metabolic advantages (Renaud and De Lorgeril 1992). It has also been postulated that phenolic substances or bioflavenoids in red wine protect LDL from oxidative damage thus reducing the risk of atherosclerotic disease (Frankel et al 1993). Although the precise mechanism by which moderate alcohol consumption protects against coronary heart disease is unclear, it is a plausible explanation of the lower cardiovascular mortality rates among men from Southern Europe but is less likely so for women because of their lower consumption relative to Australian-born women.

Western, Northern and Eastern Europeans

Age-standardised mortality rates for Germany, and the Netherlands, the most common sources of immigrants from Western Europe, were marginally lower in 1987-89 than those for Australian-born men and women but only the difference for women from the Netherlands was statistically significant. The only significant beneficial differential in risk factor levels for male immigrants from Western Europe was a higher prevalence rate of light alcohol intake. Detrimental risk factor differentials were higher total and LDL cholesterol, and higher TC/HDL. The only significant differential for women was a higher smoking prevalence rate.

Eastern European male immigrants had a significantly higher systolic blood pressure and body mass index, and were less likely to exercise during leisure-time than Australian-born men. Their prevalence rate of light alcohol intake was higher. The only significant differential for women from Eastern Europe was related to a higher prevalence of physical inactivity. Immigrants from Poland, the most common source of immigrants from these regions, were one of the few groups to have had a significantly higher SMR for cardiovascular disease compared with Australian-born men and women (Table 6.2).

Immigrants from the Middle East and North Africa

The men had a detrimental risk factor profile comprising a relatively low HDL cholesterol, high TC/HDL, high body mass index, high smoking prevalence rate, low prevalence rate for light alcohol intake and high prevalence rate of leisure-time physical inactivity compared with

Australian-born men. Despite this, their systolic blood pressure was significantly lower. The cardiovascular SMRs for Lebanon and Egypt were lower than that for Australian-born men (Table 6.2). The women were characterised by disadvantageous levels of HDL cholesterol, triglyceride, TC/HDL, body mass index, light alcohol intake and physical inactivity. As for the men, their systolic and diastolic blood pressures were lower. The SMRs for women from Lebanon and Egypt were higher than for Australian women although the differences were not statistically significant.

Asians

Although basically similar, the risk factor profiles of immigrants from Southeast Asia, and immigrants from Other Asia differed in important aspects. For men, both immigrant groups had lower systolic blood pressure and lower body mass index but only immigrants from Southeast Asia had a lower prevalence rate of light alcohol intake (due to a higher prevalence rate of non-drinkers). Men from Southeast Asia also had a significantly higher rate of leisure-time inactivity. Asian women tended to have lower body mass index, lower smoking prevalence rates, lower prevalence rates of light alcohol intake and lower rates of physical inactivity. Women from Southeast Asia had lower systolic blood pressure and higher triglyceride. Women from Other Asia had lower HDL cholesterol and higher TC/HDL.

Cardiovascular SMRs for immigrants from Southeast Asia (Vietnam, Malaysia, Philippines) and from Other Asia (India, China, Hong Kong and Macao) were all lower than those for Australian-born men and women (Table 6.2). With the exception of relatively higher prevalence rates of leisure-time physical inactivity, this is consistent with their generally advantageous risk factor profiles. The data suggest that blood lipids have played little part in their favourable cardiovascular SMR differential as the lipid profiles of Asian immigrants were generally similar to those of Australian-born men and women.

New Zealanders

Immigrants from New Zealand had a very similar risk factor profile to that of Australian-born men and women. The data suggest that their alcohol consumption pattern may be different to that of Australian-born men and women (Table 6.7). Their TC/HDL ratios were lower but did not reach statistical significance (Table 6.6). At the beginning of the 1980s, immigrants from New Zealand had relatively lower cardiovascular SMRs but this has been increasing to a point where their SMRs were not statistically different to those for Australian-born men and women in 1987-89 (Table 6.2).

However, this does not necessarily reflect a deleterious change in risk factor levels. The increase could reflect the higher proportion of Maoris in the migration stream from New Zealand in recent years.(Young 1992b)

Period of residence

Generally, analyses which have examined the influence of period of residence in Australia on mortality or on risk factor levels have not taken into account the confounding effect of the marked variation in birthplace composition of immigrants over time. This present analysis attempts to allow for this by examining changes in risk factor levels with duration of residence for broad immigrant groups and also by standardising across immigrant groups. Caution is necessary in interpretation however. Evidence of acculturation within a particular immigrant group may simply reflect the fact that longer stay immigrant residents had a different risk factor profile on arrival to Australia than those who arrived more recently. Lack of evidence may indicate that the period of residence dichotomy used in this analysis is insensitive to the acculturation effect. Risk factor differentials which existed at the time of immigration may have been moderated by general environmental factors operating immediately after settlement in Australia and a finer classification of period in Australia may be required to detect this effect.

Nevertheless, the results suggest that acculturation has had minimal effect on many of the traditional risk factors for cardiovascular disease. There is evidence that immigrants have generally had lower blood pressure on arrival to Australia which, after adjusting for age, increased as their time in Australia increased. There is also evidence that light alcohol intake became more prevalent as length of stay increased, particularly among Asian immigrants and women from Europe. There is also evidence of positive increases in exercise during leisure-time. There is no evidence of any marked changes in the blood lipid profile of immigrants with increasing stay in Australia nor any real change in smoking prevalence. Mortality analysis indicates increasing SMRs for cardiovascular disease with period of residence in Australia for most immigrant groups (Young 1986).

Methodological issues

Although difficult to quantify, it is important to identify aspects of the survey methods that have influenced the analysis. Despite the inclusion of an explanation in eleven languages with the letter of invitation in 1989, immigrants represented 35% of the target population but only 27% of respondents (Risk Factor Prevalence Study Management Committee 1990).

Using the electoral roll as the sampling frame will have led to immigrants being under-represented in the sample, particularly recent immigrants. It is also possible that the response rate among immigrants was lower than among native-born Australians. Markedly different response rates between immigrant groups would have confounded the analysis if propensity to respond was associated with risk factor characteristics.

Since no oversampling strategies were adopted to increase the number of immigrants in each survey, it was necessary to combine the three surveys so that tests for differentials had adequate statistical power. The minimal detectable difference, given a two-sided significance level of 0.05 and a power of 80%, was around 5% of the Australian mean for most risk factors, which suggests that sample sizes were adequate for the analysis.

The adjustment for age assumed a linear relationship between age and the dependent variable and that the same relationship held for each immigrant group and each survey. A more complex model would be difficult to justify given the relatively small sample size for some immigrant groups. Also, to the extent that immigrant groups congregate in different cities, adjusting for the city effect may also have partially adjusted for immigrant group risk factor differentials. The aggregation of individual countries into broad regions may have masked important differences.

Regarding the body mass analysis, perhaps the issue is whether body mass index is an appropriate indicator of cardiovascular risk across such a diverse range of immigrant groups. Several measures of body fat distribution are used in the literature and they are known to have different properties (Björntorp 1988; Himes et al. 1991; Mueller et al. 1991). The 1989 survey also collected waist and hip circumference and the result of analysing waist to hip ratio were similar to those based on body mass index with the following exceptions. Women from Asia had negative differentials for body mass index but positive ones for waist to hip ratio, and women from Western Europe had a higher mean waist to hip ratio but not body mass index, indicating different body shapes.

There is debate about the role of alcohol consumption in cardiovascular disease and whether light to moderate drinking is protective. A recent review of the evidence concluded that two drinks a day was associated with no cardiovascular harm and may be protective against coronary heart disease, and that above two drinks a day there was evidence of harm, biological as well as social (Marmot and Brunner 1991). A recent analysis of MRFIT data suggests that, in men, the protective effect may be related to

HDL cholesterol levels, which increases with alcohol consumption (Suh et al. 1992). The indicator in this analysis, `light alcohol intake', was based on the definition of responsible levels of alcohol consumption recommended in Australia (National Health and Medical Research Council 1987b).

There is no generally accepted instrument for the measurement of physical activity in population surveys. Studies have tended to focus on leisure-time activity, but recent evidence suggests that both leisure-time exercise and exercise at work reduce coronary heart disease risk, although their relative contribution is unclear (Salonen et al. 1988), and may well differ for different immigrant groups (Zimmet et al. 1991). Regarding leisure-time physical activity, recent years have seen a shift in emphasis away from the more vigorous aerobic exercise towards the health benefits of less vigorous exercise (Blair et al. 1989; De Busk et al. 1990; Oldenburg et al. 1991; National Heart Foundation of Australia 1991a). The 1991 Heart Week in Australia emphasised the benefits of walking for exercise (National Heart Foundation of Australia 1991c). The indicator used in this analysis, no leisure-time exercise of any kind, implicitly considered vigorous exercise, less vigorous exercise and walking all to be valued forms of exercise to reduce coronary heart disease risk.

6.5 Concluding remarks

This analysis has identified differences in risk factor levels among Australia's immigrants when compared with their Australian-born counterparts. However, it would appear that the lower SMRs from cardiovascular disease which characterise immigrants in Australia cannot be explained solely on the basis of their profiles of risk factors which are commonly accepted as determinants of cardiovascular disease. Of the traditional risk factors, systolic blood pressure is the best single explanatory factor of the variation between immigrant groups in cardiovascular mortality among men, and smoking prevalence the best among women (Table 6.9). The results of a regression analysis of mean risk factor levels against standardised mortality rates were consistent with this conclusion.

One of the most interesting findings was the lack of clear differentials in blood lipids. An individual's blood lipid profile may be influenced by factors such as physical activity, alcohol consumption, use of oral contraceptives, aspects of diet such as dietary fibre and saturated fats, as well as genetic factors. These, and other factors, will combine to determine the blood lipid levels of a particular immigrant group. It seems that in Australia, such factors have largely balanced each other out and that blood lipids, including

total cholesterol, have played little part in explaining cardiovascular mortality differentials.

TABLE 6.9 *Cardiovascular mortality and risk factor profiles by immigrant group*

Immigrant group	SMR	SBP	DBP	BMI	TC	HDL	TG	LDL	TC/HDL	SMK	LTA	INA
Men												
Southeast Asia	60	+	.	+	.	.	.	.	.	.	-	-
Other Asia	62	+	.	+	.	.	.	.	.	.	.	.
Greece	62	+	.	-	.	-	.	.	.	-	.	-
Italy	67	+	.	-	+	.	.	+	.	-	+	-
The Middle East	85	+	.	-	.	-	.	.	-	-	-	-
England and Wales	92	+	+	+	.	.	.	.	.	.	+	.
Scotland and Ireland	92	.	.	.	.	.	.	.	.	-	.	-
Western Europe	93	.	.	.	-	.	.	-	-	.	+	.
Other Southern Europe	95	.	.	-	.	.	.	.	.	-	.	-
Other countries	100	.	.	.	.	.	.	.	.	.	.	.
New Zealand	109	.	.	.	.	.	.	.	.	.	.	.
Eastern Europe	124	-	.	-	.	.	.	.	.	.	+	-
Women												
Southeast Asia	50	+	.	+	.	.	.	.	.	+	-	-
Greece	55	.	.	-	.	-	.	.	-	+	-	-
Italy	68	.	.	-	+	-	.	+	.	+	-	-
Other Asia	70	.	.	+	.	-	.	.	-	+	-	-
Western Europe	87	.	.	.	.	.	.	.	.	-	.	.
England and Wales	91	+	+	+	.	.	.	.	.	.	+	.
Scotland and Ireland	91	.	.	.	.	.	.	.	.	-	.	.
Other Southern Europe	100	.	.	-	.	-	.	.	.	.	-	-
Other countries	102	.	.	.	.	.	.	.	.	.	.	.
New Zealand	105	.	.	.	.	.	.	.	.	.	.	.
Eastern Europe	117	.	.	.	.	.	.	.	.	.	.	-
The Middle East	120	+	+	-	.	-	-	.	-	.	-	-

Note: The table shows which comparisons with the Australian-born were statistically significant ($p<.05$), after adjusting for age and survey centre. The comparisons have been classified as beneficial (+) or detrimental (-). Beneficial risk factor levels were taken as lower SBP, DBP, TC, TG, LDL, TC/HDL, BMI, SMK and INA (physical inactivity), and higher HDL and LTA (light alcohol intake). Immigrant groups have been ordered by their SMRs for cardiovascular disease 1987-89 (derived from Young 1992b).

The analysis also found that, after adjusting for the ageing effect, systolic blood pressure among immigrants increases with length of stay in Australia. Body mass index increases among Asian immigrants despite increased participation in recreational exercise with longer length of stay. There is no evidence that blood lipid profiles are affected by the process of acculturation.

The relationship between risk factor and mortality differentials for immigrant groups is complex, however, and a full explanation is likely to involve interaction between social, economic, cultural, environmental, biological and genetic factors (Polednak 1989), as well as factors relating specifically to the migration process. It may well be that the lead time between exposure to major coronary risk factors and subsequent effects on mortality also varies between immigrant groups.

Chapter Seven

Socioeconomic inequalities in cardiovascular mortality over time

7.1 Introduction

At the time that mortality from coronary heart disease peaked in Australia (the late 1960s), there was an inverse relationship between socioeconomic status and coronary heart disease mortality (in men) and evidence suggests that mortality differentials widened during the following decade while mortality declined (Gibberd et al. 1984; Dobson et al. 1985; Taylor et al. 1983). Risk factor levels were found to be consistent with mortality differentials (among men) (Dobson et al. 1985). This was analogous to the situation in Britain (Marmot 1989) and the United States (Wing et al. 1987; Feldman et al. 1989), except that in Britain the decline in coronary heart disease mortality began later. Trends in inequalities in mortality have not received the same attention in Australia as in Britain and the United States and the analyses of Australian data for the 1970s remain the latest available.

This chapter examines trends and inequalities in coronary heart disease and stroke mortality in Australia over the period 1979 to 1993 across broad occupation groups and compares the findings with movements in major risk factors for cardiovascular disease, namely high blood pressure, high blood cholesterol and smoking. Occupation has been chosen as the indicator of socioeconomic status because it is common to the death registration and risk factor databases. Several questions are addressed. First, have the mortality inequalities observed in Australia in the 1970s persisted into the eighties and nineties? Second, has the gap continued to widen? Third, are the inequalities in mortality consistent with those in risk factors? Fourth, have those occupation groups which improved their risk factor profiles the most experienced greater improvements in mortality? Finally, can occupational inequalities in risk factors be explained by differences in educational attainment? The answers to such questions are important for assessing the impact of past health initiatives in Australia and for guiding future directions.

7.2 Trends in mortality inequalities

During the study period 1979 to 1993, deaths were coded according to the Ninth Revision of the International Classification of Diseases (ICD9). Age-standardised death rates were calculated for coronary heart disease (ICD9 410-414) and stroke (ICD9 430-438) by occupation group by single year. The analysis was conducted for men only because the quality of the occupation data on the death certificate for women is inadequate over much of the time series. The analysis was restricted to ages 25-64 because death from coronary heart disease or stroke is rare before the age of 25 and after the age of 64 most men are not in the labour force. Also, occupation has only been collected on the death certificate beyond age 64 since 1990. The age-standardised rates were based on age groups 25-34, 35-44, 45-54, 55-59, and 60-64 years, using the direct method and using employed males at the 1986 census as the standard population. Estimates of the male population at risk were taken from the Australian Bureau of Statistics' monthly labour force survey which uses multi-stage area sampling of 29,000 private dwellings and a list sample of non-private dwellings. The population distribution of the nearest census was used as a benchmark in the estimation process.

Two systems were used to code occupation during the period 1979 to 1993, the Classification and Classified List of Occupations (CCLO) and the Australian Standard Classification of Occupations (ASCO). ASCO has a more even distribution and less major groups than CCLO (Table 7.1). ASCO was introduced into the labour force series in 1986 and into the mortality data collection in 1990, resulting in common coding systems from 1979 to 1985 (CCLO), and from 1990 to 1993 (ASCO), but different systems for the period 1986 to 1989. It is not feasible to develop a simple code-for-code mapping between the two classifications because CCLO groups occupations according to industrial sector whereas ASCO is a skill-based classification. Because the two classifications are fundamentally different, the only mapping possible is a one-to-many mapping based on probabilities. Accordingly, a quantitative relationship has been derived from a link file developed by the Australian Bureau of Statistics based on dual coding of a 5% sample of responses from the 1986 census (Australian Bureau of Statistics 1988a, 1988b). This file has been used to derive a matrix of estimates, ASCO by CCLO at major group level, for males aged 25-64. The matrix was used to convert the labour force estimates for the period 1986 to 1989, from ASCO to CCLO, for compatibility with the mortality data.

TABLE 7.1 *Distribution of labour force based on ASCO and CCLO coding systems for men aged 25-64*

Code	Major group description	per cent
ASCO coding system		
1	Managers and Administrators	16.6
2	Professionals	13.4
3	Para-Professionals	6.8
4	Tradespersons	20.7
5	Clerks	7.3
6	Salespersons and Personal Service Workers	7.5
7	Plant and machine Operators, and Drivers	12.4
8	Labourers and Related workers	13.7
9	Occupations inadequately described or not stated	1.7
CCLO coding system		
0	Professional, technical & related workers	14.7
1	Administrative, executive and managerial workers	12.2
2	Clerical workers	7.3
3	Sales workers	6.4
4	Farmers, fishermen, hunters, timber getters & related workers	6.9
5	Miners, quarrymen & related workers	1.0
6	Workers in transport and communication	7.7
7/8	Tradesmen, production-process workers & labourers, nec	36.2
9	Service, sport & recreation workers	6.0
10	Members of armed services	1.4
11	Occupations inadequately described or not stated	0.3
Estimated total labour force for men aged 25-64		3,134,138

(a) Derived from a 5% dual coding of the 1986 Census of Population and Housing

The matrix was also used to determine three broad occupation groups on which the analysis was based. For ease of reference, these groups have been labelled 'professional', 'clerical/sales' and 'manual' (Table 7.2). Broad groupings were chosen to protect against undue variation resulting from small numbers and to moderate the effect of the change in coding system.

TABLE 7.2 *Definition of broad occupation groups*

Data source	1979-85	1986-89	1990-93
Mortality data			
Coding system	*CCLO*	*CCLO*	*ASCO*
Professional	0,1,4	0,1,4	1,2,3,9001,9005
Clerical/sales	2,3	2,3	5,6
Manual	5,6,7/8,9	5,6,7/8,9	4,7,8,9002,9004
Armed forces (excluded)	10	10	9003
Not in workforce (excluded)	960,970,980, 1000, 9999	960,970,980, 1000, 9999	1 or 2 in 10's col. 9013-9720
Insufficient information	950	950	9006,9800
Not stated	990	990	9900
Labour force data			
Coding system	*CCLO*	*ASCO*[a]	*ASCO*
Professional	0,1,4	1,2,3	1,2,3
Clerical/sales	2,3	5,6	5,6
Manual	5,6,7/8,9	4,7,8	4,7,8

(a) CCLO estimates were derived from ASCO estimates using conversion matrix

Deaths which had 'occupation' coded as 'insufficient information' or 'not stated' varied over time between 3% and 12% of total deaths (greater for ASCO) and were distributed proportionally across all other categories. Deaths for members of the permanent defence forces and deaths for men

'not in the workforce' were excluded from the death registration data to be consistent with coverage of the labour force survey. 'Not in the workforce' included the retired, invalid pensioners, other pensioners, home duties, and the unemployed.

Table 7.3 depicts the degree of commonality between the composition of each broad occupation group as defined by ASCO and by CCLO. Eighty-two per cent of the CCLO professional group were common to the professional group under ASCO. Equivalent estimates for the clerical/sales and manual groups were 73% and 83% respectively.

TABLE 7.3 ***Association between broad occupation groups based on CCLO and ASCO, men aged 25-64 (a)***

	ASCO				
CCLO	Professional	Clerical/sales	Manual	Other	Total
Professional	82.0	6.4	10.3	1.2	100.0
Clerical/sales	21.4	73.0	3.9	1.7	100.0
Manual	10.8	4.8	82.9	1.6	100.0
Other	39.6	12.1	34.9	13.4	100.0
Total	36.8	14.8	46.8	1.7	100.0

	ASCO				
CCLO	Professional	Clerical/sales	Manual	Other	Total
Professional	75.4	14.7	7.5	24.0	33.8
Clerical/sales	8.0	67.6	1.2	13.5	13.7
Manual	14.9	16.4	90.2	49.3	50.9
Other	1.8	1.4	1.2	13.1	1.7
Total	100.0	100.0	100.0	100.0	100.0

(a) Estimates derived from 5% dual coding of 1986 Census of Population and Housing

Annual rates of change in mortality were estimated by fitting a log linear model with Poisson error distribution and the natural log of population size as an 'offset' (Breslow and Day 1987; Valkonen 1989a). For age-standardised death rates the model may be expressed as:

$$\log_e(D_{kt}) = \log_e(N) + \text{constant} + a_k t$$

where t = the year of registration of death, k = occupation group, D_{kt} = the expected number of deaths in year t for occupation group k, N = the total standard population, and a_k = the estimated rate of increase or decrease in mortality for occupation group k. The annual rate of change was derived as $\exp(a_k) - 1$.

Trends in mortality from coronary heart disease and stroke are shown in Figure 7.1 for each occupation group. For the period 1979-1993, death rates for coronary heart disease and stroke were invariably highest among manual occupations, independent of the coding system used for occupation.

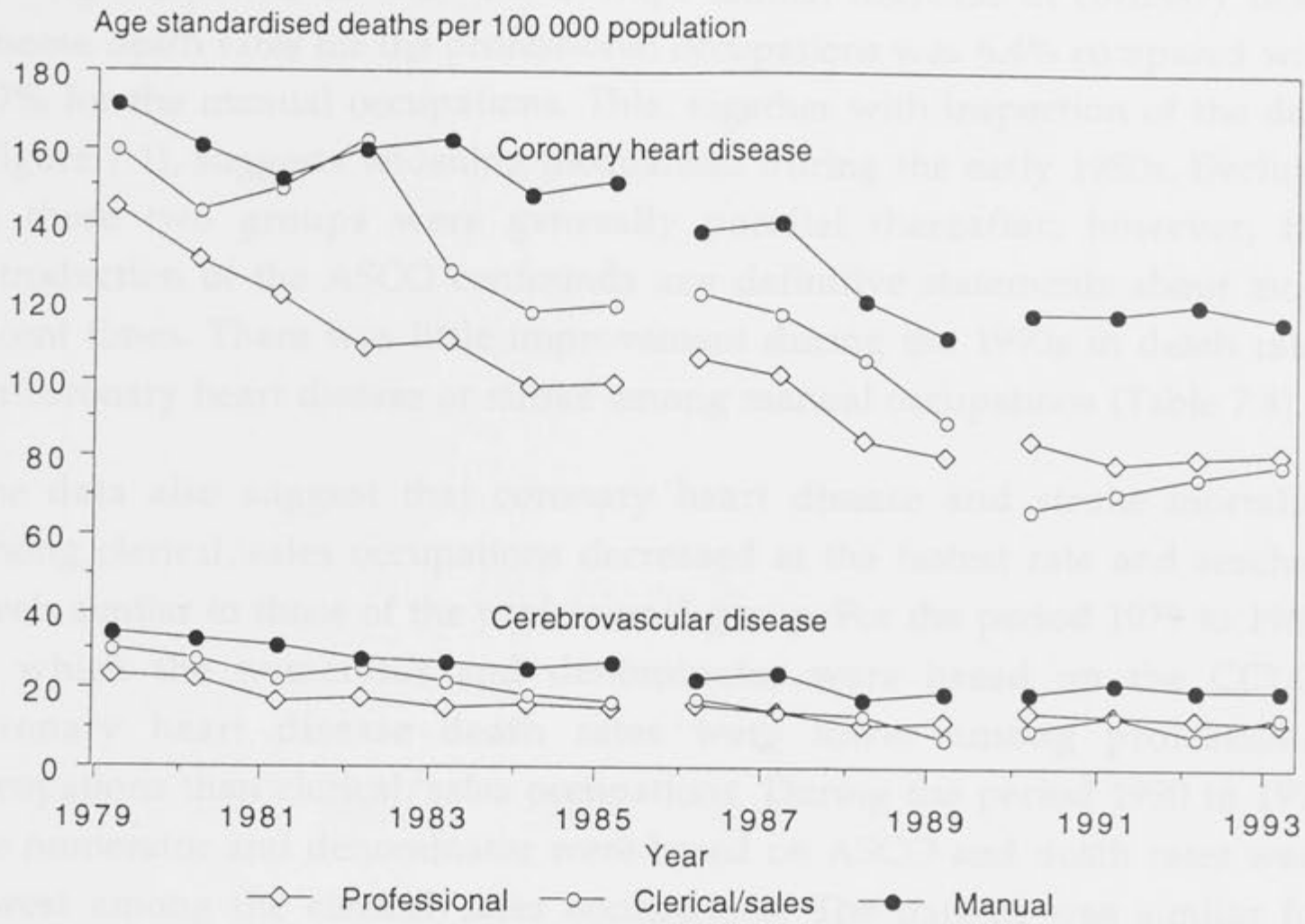

FIGURE 7.1 ***Death rates from coronary heart disease and cerebrovascular disease, Australian men aged 25-64 by occupation group, 1979-1993.*** *[breaks indicate changes in occupation coding systems; see text]*

On average, men in manual occupations were 35% more likely to die from coronary heart disease than men in professional occupations, and 60% more likely to die from stroke (Table 7.4).

TABLE 7.4 ***Annual death rate and change for coronary heart disease and stroke, occupation group by period, men aged 25-64***

	Average annual death rate (a)			Average annual change (%)		
Occupation group	1979-85	1986-89	1990-93	1979-85	1986-89	1990-93
			Coronary heart disease			
Professional	115.4	94.3	81.0	-6.4#	-9.3#	-1.2
Clerical/sales	139.7	110.0	72.1	-4.9#	-9.8#	+5.9#
Manual	157.7	126.3	117.5	-1.7#	-7.7#	-0.4
Mortality ratio (b)	1.37#	1.34#	1.45#			
95% CI	(1.33,1.40)	(1.29,1.39)	(1.40,1.51)			
			Stroke			
Professional	18.1	13.6	13.0	-7.4#	-11.7#	-6.2#
Clerical/sales	23.4	13.5	11.3	-8.7#	-21.9#	+3.5
Manual	29.5	20.9	20.5	-5.0#	-8.6#	+0.5
Mortality ratio (b)	1.63#	1.53#	1.58#			
95% CI	(1.53, 1.73)	(1.40,1.68)	(1.44,1.73)			

(a) Age standardised, per 100,000 population. (b) Ratio of Manual to Professional.
95% confidence intervals, in brackets, are calculated according to Boyle and Parkin 1991. # $p<0.01$.

It is clear that each occupation group experienced significant falls in coronary heart disease (and stroke) mortality during the 1980s but that the socioeconomic inequalities of the 1970s persisted into the early nineties.

During the period 1979-85, the average annual decrease in coronary heart disease death rates for the professional occupations was 6.4% compared with 1.7% for the manual occupations. This, together with inspection of the data (Figure 7.1), suggests widening inequalities during the early 1980s. Declines in these two groups were generally parallel thereafter; however, the introduction of the ASCO confounds any definitive statements about more recent times. There was little improvement during the 1990s in death rates for coronary heart disease or stroke among manual occupations (Table 7.4).

The data also suggest that coronary heart disease and stroke mortality among clerical/sales occupations decreased at the fastest rate and reached levels similar to those of the professional group. For the period 1979 to 1989, in which the numerator and denominator were based on the CCLO, coronary heart disease death rates were lower among professional occupations than clerical/sales occupations. During the period 1990 to 1993 the numerator and denominator were based on ASCO and death rates were lowest among the clerical/sales occupations. The pattern was similar for stroke except that rates for the clerical/sales occupations and the professional group become very close during the period 1986 to 1989. Estimates from 1990 reflect the introduction of ASCO, however the time series for stroke mortality suggest that the relative position of the clerical/sales group began to improve during the early 1980s, before the ASCO coding system was introduced. To investigate further, the transition probability matrix[1] was used to convert both 1990-93 deaths and labour force data from ASCO to CCLO and the rates recalculated, but this did not alter the reversal in mortality ranking for the professional and clerical/sales groups which occurred between 1986-89 and 1990-93. The reversal will reflect, to an unknown extent, the different composition of the two groups under the different classification systems.

This phenomenon was examined further by repeating the analysis for the census years, 1981, 1986, 1989, using equivalent methodology but substituting census estimates of the employed population by occupation group in place of labour force survey estimates. The results were consistent with the trends and inequalities identified using annual estimates from the labour force survey (Figure 7.2). In particular, death rates were highest among manual occupations for each of the three censuses, and were lowest among clerical/sales occupations in 1991.

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[1] At the major group level, as described in Table 7.1.

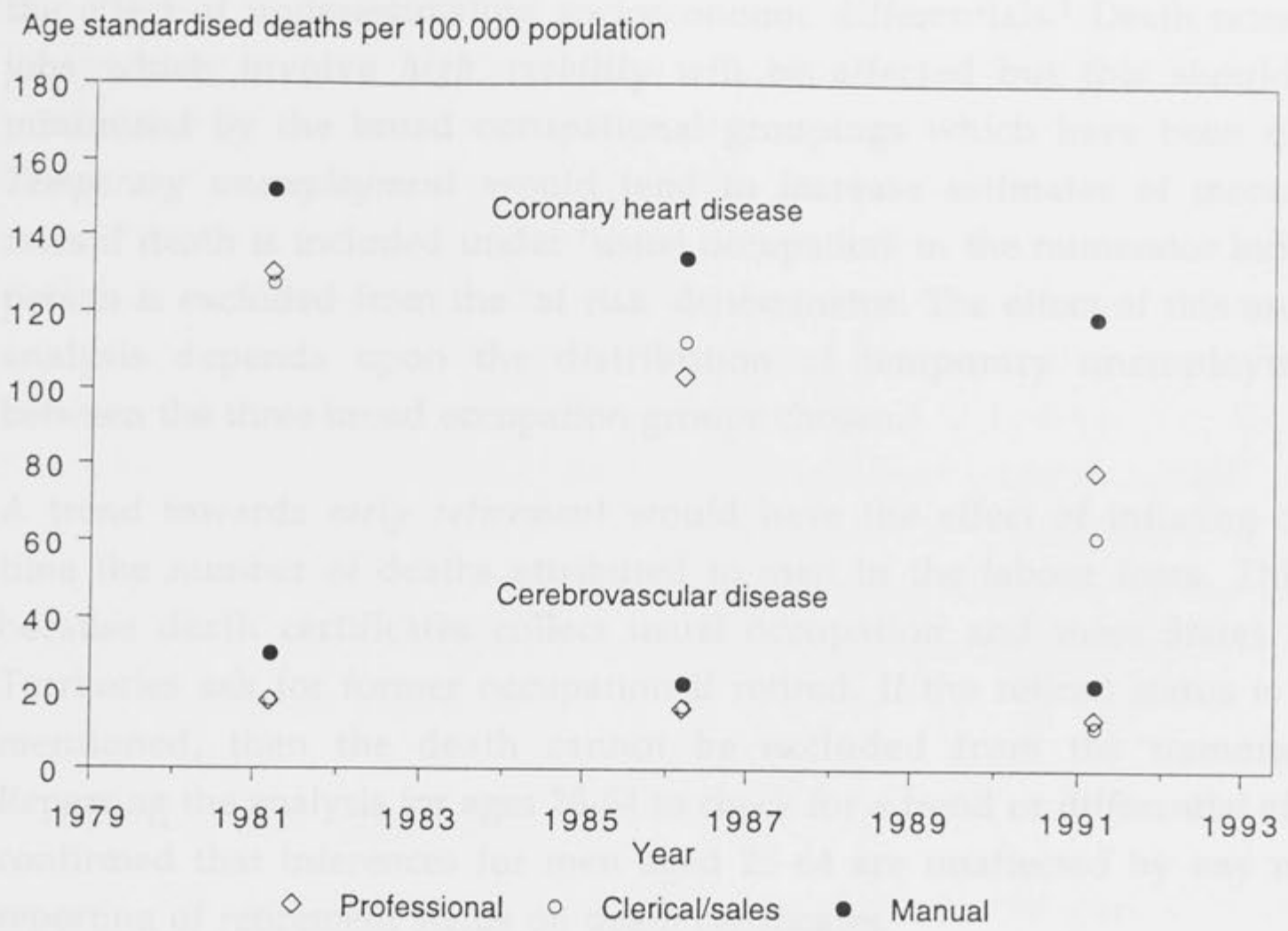

FIGURE 7.2 *Death rates from coronary heart disease and cerebrovascular disease, Australian men aged 25-64 by occupation group, census years 1981, 1986, 1991*

7.3 Potential biases

Several potential sources of bias in the mortality analysis need to be addressed, especially those relating to numerator/denominator inconsistencies arising from the need to use two separate data sources to calculate rates (Taylor et al. 1983; McMichael and Hartshorne 1980). Death certificates are coded for 'usual or last occupation' and this information is supplied by a relative or other person acquainted with the deceased, or by an official of the institution where the death occurred. The labour force survey collects occupation last week, or 'current occupation', by a trained interviewer. This effect of this inconsistency was minimised by restricting the analysis to men aged 25-64 and by confining attention to three broad occupation groups. Care was taken to make coverage of the numerator and denominator as compatible as possible.

A tendency to report *socially desirable* occupations on the death certificate at the expense of less prestigious current or recent jobs would tend to overestimate mortality in higher socioeconomic occupations, thus moderating socioeconomic differentials. A tendency to move towards a lower socioeconomic occupation towards *retirement* would likewise have

the effect of underestimating socioeconomic differentials.[1] Death rates for jobs which involve *high mobility* will be affected but this should be minimised by the broad occupational groupings which have been used. *Temporary unemployment* would tend to increase estimates of mortality rates if death is included under `usual occupation' in the numerator but the person is excluded from the `at risk' denominator. The effect of this on the analysis depends upon the distribution of temporary unemployment between the three broad occupation groups chosen.

A trend towards *early retirement* would have the effect of inflating over time the number of deaths attributed to men in the labour force. This is because death certificates collect usual occupation and most States and Territories ask for former occupation if retired. If the retired status is not mentioned, then the death cannot be excluded from the numerator. Repeating the analysis for ages 25-54 to check for a trend or differential effect confirmed that inferences for men aged 25-64 are unaffected by any non-reporting of retirement status on death certificates.

All deaths for members of the permanent *defence forces* were readily identifiable under CCLO and were excluded. Under ASCO, however, such deaths were allocated to the appropriate skill-based category and not all could be identified. It was estimated that approximately four deaths per annum were included in estimates for 1990-93 which should have been excluded, and the effect of this would have been negligible.

Finally, mortality data relate to year of registration of death, not year of occurrence, and usually 5-6 per cent of deaths occurring in one year are not registered until the following year or later. In 1984 there were abnormal delays in the registration process in one State which had the effect of displacing approximately 2000 registrations from 1984 to 1985 (1.7% of all deaths in Australia). This will have had little effect on the estimates of trends or inequalities.

On balance, the methodology adopted in this paper seems to have produced sensible estimates of mortality trends and conservative estimates of

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[1] The death certificate collects 'usual or last occupation' and is reported by a relative whereas the labour force collects current occupation by a trained interviewer. To the extent that the death certificate records lifetime occupation (say professional) whereas the labour force collects occupation prior to death (say manual), the death would be counted in the numerator (but not denominator) for the professional group (producing an overestimate) and in the denominator (but not numerator) for the manual group (producing an underestimate). The net effect would be to moderate the socioeconomic differentials in death rates.

inequalities. The same classification of diseases was used throughout the study period and the quality of official mortality data for ICD9 410-414 has been validated in Australia and found to be reasonably accurate and reliable (Dobson et al. 1983; Sexton et al. 1992). The careful formation of broad occupation groupings minimised the effect of the change from CCLO to ASCO but will have had a moderating effect on socioeconomic differences. Also, both occupation coding systems produce heterogeneous occupational groups with respect to socioeconomic status, ASCO less so than CCLO, which has the effect of diluting statistical associations (Najman 1988; Turrell et al. 1994). The supplementary census analysis for 1981, 1986 and 1991 supported the findings of the time series analysis based on labour force statistics. It is concluded that the mortality inequalities are unlikely to be the product of statistical artefacts.

7.4 Possible explanations

How is this association between occupation group and mortality from coronary heart disease and from stroke best explained? Differences in ethnic composition have the potential to be a partial explanation of the mortality inequalities observed, given that Australians born overseas are known to have lower death rates from coronary heart disease and stroke than people born in Australia (Young 1986). In fact, the proportion of overseas born is higher among the manual group than the professional or clerical/sales group and the mortality inequalities would have been greater had adjustment been made for ethnic composition.

Inequalities and trends in mortality rates should reflect, to some extent, inequalities and trends in risk factors at a previous time. This hypothesis can be examined by using data from the Risk Factor Prevalence Study. The Study collected self-reported occupation using the CCLO list of major groups in 1980 and 1983 , and the ASCO coding system in 1989. The same broad occupation classifications were used for the risk factor analysis as for the mortality analysis and the analysis was confined to employed males in the age range 25-64 years. Sample sizes averaged 1272 for the professional group, 414 for the clerical/sales group and 1083 for the manual group. The analysis was conducted for blood pressure, total cholesterol and cigarette smoking, factors which are believed to be of greatest value in predicting the risk of major ischaemic heart disease (Shaper et al. 1985).

For blood pressure and total cholesterol, comparisons between occupation groups and associations over time were examined by analysis of

covariance. The model included survey year, age, survey centre and birthplace (Australian-born or overseas-born) as independent variables in order to allow for differences in the composition of participants over time. An interaction term between occupation group and year of survey was included to allow for different trends in different occupation groups. Multiple logistic regression was used to examine changes over time in the proportion of smokers in each occupation group using the same independent variables as for continuous variables. Trends in prevalence odds ratios and their 95% confidence limits were derived from the maximum likelihood parameter estimates of the model (Table 7.5).

TABLE 7.5 ***Trends and inequalities in risk factors by occupation group, men aged 25-64.***

	1980	1983	1989	1989/1980	
Occupation group	Crude estimates			Trends	
	Systolic blood pressure (mmHg)				
Professional	130.9	129.3	127.8	-3.2#	(-4.5,-1.9)
Clerical/sales	133.3	131.7	129.5	-4.8#	(-7.0,-2.5)
Manual	134.7	135.4	129.5	-5.0#	(-6.4,-3.6)
Inequalities	+2.8#	+4.9#	+0.9		
	(-4.1,-1.4)	(3.7,6.1)	(-0.4,2.3)		
	Hypertension (%)				
Professional	23.6	15.6	14.7	0.55#	(0.43,0.68)
Clerical/sales	26.9	21.6	18.0	0.50#	(0.35,0.71)
Manual	25.9	25.0	18.5	0.66#	(0.52,0.83)
Inequalities	1.01	1.61#	1.22		
	(0.72,1.42)	(1.32,1.96)	(0.96,1.55)		
	Total cholesterol (mmol/L)				
Professional	5.68	5.63	5.59	-0.08	(-0.17, 0.00)
Clerical/sales	5.60	5.75	5.79	+0.15*	(0.00, 0.30)
Manual	5.72	5.60	5.58	-0.12*	(-0.22,-0.03)
Inequalities	-0.03	-0.11#	-0.07		
	(-0.12,0.07)	(-0.19,-0.03)	(-0.16,0.03)		
	Raised cholesterol (%)				
Professional	20.9	19.2	19.8	0.95	(0.77,1.17)
Clerical/sales	18.0	22.3	24.4	1.39	(0.97,1.98)
Manual	20.8	18.0	19.2	0.94	(0.74,1.18)
Inequalities	0.88	0.79	0.87		
	(0.71,1.10)	(0.65,0.97)	(0.69,1.09)		
	Current smokers (%)				
Professional	28.0	25.9	18.4	0.58#	(0.48,0.71)
Clerical/sales	37.1	39.2	31.2	0.78	(0.58,1.05)
Manual	45.5	41.1	33.1	0.60#	(0.50,0.73)
Inequalities	2.12#	1.99#	2.19#		
	(1.76,2.55)	(1.70,2.34)	(1.79,2.67)		

Trends are calculated as odds ratios (1989/1980) for categorical variables and differences (1989-1980) for continuous variables; similarly inequalities are calculated as odds ratios (Manual/Professional) or differences (Manual-Professional). Both trends and inequalities are adjusted for age and survey design factors, with 95% confidence limits in brackets. * $p<0.05$, # $p<0.01$.

Mean systolic blood pressure and the prevalence of hypertension were lower among professional occupations, but all occupation groups experienced a decline over the period 1980-89 and the differential declined (Table 7.5). Differentials and trends for total cholesterol showed no patterns favouring any particular occupation group. Smoking prevalence showed a clear negative association with socioeconomic status and prevalence declined over time in each occupation group.

These same risk factors have been used to quantify the 5-year risk of a coronary event, a fatal or non-fatal heart attack, for each individual using the multiple logistic function (the Dundee risk equation) derived from men aged 40-59 in the United Kingdom Heart Disease Prevention Project (Tunstall-Pedoe 1991). For valid prediction of risk in one population, based on an equation from another, various assumptions need to be satisfied (Chambless et al. 1990). In this application, care was taken to ensure the definition of variables was consistent and to apply the equation to the same age-sex group. In addition, the analysis focused on relative risk and trend estimates rather than absolute risk levels. The 5-year risk (p) was based on the three major modifiable risk factors and calculated as

$$\frac{1}{1+e^{-(a+b_1x_1+b_2x_2+b_3x_3)}}$$

where $a = -6.8624$

$b_1 = 0.010543$ x_1 = systolic blood pressure (mmHg)

$b_2 = 0.3627$ x_2 = cholesterol (mmol/l)

$b_3 = 1.00$ x_3 = smoking code based on cigarettes per day

1-4/day	= 0.406
5-9/day	= 0.406 + 0.0813 x (cigarettes - 5)
10-29/day	= 0.8125 + 0.0312 x (cigarettes - 10)
≥ 30/day	= 1.437

Inequalities and trends in the population risk of major coronary heart disease were examined by modelling log (p) with survey year, age, survey centre and birthplace (Australian-born or overseas-born) as independent variables. As a supplementary analysis, educational attainment was added into the model to examine its role in explaining occupational inequalities in 5-year risk.

For men aged 40-59, the 5-year risk of a coronary event was lowest among professional occupations, highest among manual occupations and both groups experienced significant declines in risk over the 1980s (Table 7.6). The 5-year risk of a fatal or non-fatal coronary event was 30% higher among manual than professional occupations. Improvements in coronary risk were

approximately equal in both these groups, as were improvements in mortality. The clerical/sales group experienced the greatest falls in mortality but little improvement in overall 5-year risk (Table 7.6). However, the trend estimates will have been influenced by the change in occupational coding system and should be interpreted with caution.

TABLE 7.6 *Trends and inequalities in coronary heart disease mortality and 5-year risk of a coronary event by occupation group, men aged 40-59*

Occupation	1980	1983	1989	1989/1980	
	5-year risk of coronary event (a)				
Professional	0.049	0.045	0.041	0.84#	(0.77,0.91)
Clerical/sales	0.053	0.056	0.052	0.98	(0.85,1.13)
Manual	0.065	0.058	0.053	0.83#	(0.76,0.90)
Relative risk (b)	1.33#	1.30#	1.31#		
95% CI	(1.22,1.45)	(1.20,1.40)	(1.20,1.43)		
	1981-85	1984-88	1990-93	1989/1980	
	Average annual death rate (c)				
Professional	152.5	133.4	99.2	0.65#	(0.60,0.71)
Clerical/sales	190.4	156.5	91.9	0.48#	(0.42,0.55)
Manual	212.6	183.6	153.9	0.72#	(0.68,0.77)
Mortality ratio (b)	1.39#	1.38#	1.55#		
95% CI	(1.31, 1.48)	(1.29, 1.47)	(1.43, 1.68)		

(a) Adjusted for age and survey design factors.

(b) Ratio of Manual to Professional.

(c) Age standardised, per 100,000 population.

95% confidence limits, in brackets, are calculated according to Boyle and Parkin 1991.

# $p<0.01$.

Although each survey achieved a response rate very close to 75%, which is very high for surveys which include physical examination and venipuncture, it is possible that the analysis has been affected by response bias. Other studies have found that people with an adverse risk factor profile are less likely to participate in surveys such as these (Bergstrand et al. 1983; Criqui et al. 1978). If this were the case in this study, the inequalities observed would be underestimates of the true differentials, since risk factors are more prevalent in low socioeconomic groups. Overestimation of the association between socioeconomic status and risk factors would occur only if propensity to respond was associated with a better risk factor profile among the professional occupations and worse profile among the manual occupations, a phenomenon which has not been reported in other studies. There are no data available on the characteristics of nonrespondents to enable the calculation of response rates for occupation groups over time but the remarkable consistency in overall response rates offers some assurance against biased trend estimates. Characteristics of nonrespondents have been found to be relatively stable over time (Sprafka et al. 1990).

The risk factors used in this analysis are best for predicting the risk of major ischaemic heart disease (Shaper et al. 1985). However, in the present setting, risk factor measurements were taken at a single visit and will have a weaker relationship with disease outcome than have usual risk factor levels (MacMahon 1990). In addition, the measurement of socioeconomic status is subject to the limitations of the occupational classification system. The combination of these effects will lead to underestimation of the strength of the relationships between coronary risk, mortality and socioeconomic status. Despite this, and the differences in coverage between the risk factor and mortality data sources, the pattern in 5-year risk was generally consistent with subsequent mortality experience at the population level. The socioeconomic distribution of blood pressure and smoking prevalence is most likely to have contributed to the socioeconomic inequalities in the 5-year risk of a heart attack and in the subsequent mortality from coronary heart disease. Decreases in these risk factors will have contributed to the declines in 5-year risk and coronary mortality. Since total cholesterol does not appear to have contributed to either the trends or inequalities observed in mortality, the additional benefits which could be expected from reduced population levels in total cholesterol are yet to be realised.

It is possible that a differential experience of work stress may contribute to the mortality differentials. Stressors associated with physical work conditions (eg noise, heavy lifting), psychomental workload (eg high demand but low reward or insufficient control, repetitive or irregular work, job insecurity), and reward structure (eg insufficient wages, poor career opportunities) are associated with increased coronary risk and are more typical of manual workers (Siegrist et al. 1986; Siegrist 1991). Lower social support, increased social instability and poor social coping resources, believed to be typical of lower socioeconomic groups, may also be implicated (Siegrist et al. 1986).

Socioeconomic status is known to influence a person's lifestyle and their interaction with the heath system (National Health Strategy 1992) and the inverse association between socioeconomic status and coronary heart disease mortality may reflect a differential effect for prevention and medical care. In chapter 8 it is shown that risk factor levels and educational attainment are related. Adjusting the occupational inequalities in 5-year risk for educational attainment only partially explained them which suggests that education and occupation are both important influences on risk factors levels among men. This is consistent with the hypothesis that occupation and education measure different but related dimensions of socioeconomic

status (Turrell et al 1991). In the current context, occupation is likely to indicate exposure to particular physical or psychosocial factors, while educational attainment is likely to indicate knowledge and receptiveness to health education messages.

It may be that coronary heart disease in middle age is linked with poverty and adverse living conditions in the early stages of life; that coronary risk is determined by deprivation in fetal and early infant life, and that biological structures are programmed at this time which endure into adulthood (Barker 1992; Barker and Osmond 1992). Reviews of the epidemiological evidence have not found strong support for the hypothesis (Elford et al. 1991; Elford et al. 1992). Recent studies suggest that socioeconomic conditions in adulthood (Lynch et al 1994; Ben-Shlomo and Davey Smith 1991) and contemporary lifestyle changes (Baker et al. 1993) are more important predictors of coronary mortality than socioeconomic conditions in early life. This is a difficult area for epidemiological research, however, because of the complexity of the issues and the methodological problems involved (Elford et al. 1991; Baker 1994).

To answer the questions posed at the beginning of this chapter: it has been demonstrated that the mortality inequalities of the 1970s have persisted into the eighties and nineties; that the gap widened during the early 1980s but stabilised thereafter; that inequalities in mortality are consistent with those in risk factors with blood pressure and smoking particularly important; that the Professional and Manual occupation groups experienced similar improvements in cardiovascular risk and mortality; and that the occupational gradient in 5-year risk of a coronary event is only partially explained by differences in educational attainment.

Risk factor differentials are undoubtably important contributors to cardiovascular mortality differentials as it seems are socioeconomic factors, both indirectly through risk factors and directly through pathways as yet not fully understood. Behavioural and cultural, materialist and structural influences are all closely interwoven and it is difficult to disentangle their relative effects. Nevertheless, for a fuller understanding of factors leading to increased risk of cardiovascular disease it is necessary to consider life-style and biomedical risk factors in their socioeconomic and cultural context.

Chapter Eight

Socioeconomic inequalities in cardiovascular risk factors over time

8.1 Introduction

In Australia, as in other developed countries, people of lower socioeconomic status have been at greater risk of cardiovascular disease. The relationship has been documented for mortality from coronary heart disease and stroke, in men and women, and for morbidity and risk factors (McMichael and Hartshorne 1982; Taylor et al. 1983; Gibberd et al. 1984; Dobson et al. 1985; McMichael 1985; Simons et al. 1986; Siskind et al. 1987; Dobson et al. 1991c; Siskind et al. 1992; National Health Strategy 1992). There is evidence for men that the marked decline in cardiovascular mortality observed in Australia since the late 1960s was initially greater among the higher socioeconomic groups; that is, that socioeconomic inequalities were widening (Taylor et al. 1983; Gibberd et al. 1984; Dobson et al. 1985; Hardes et al. 1985). This phenomenon has been observed also in Britain and the United States (Marmot and McDowall 1986; Feldman et al. 1989).

Within developed countries, the pattern of higher rates of illness and death among people of lower socioeconomic status holds for most diseases (Marmot et al. 1987; Feinstein 1993). Reasons for this association are unclear and potential explanations include external factors and personal characteristics and behaviours of the individual. These include living and working conditions; access to health care services; cultural influences; aspects of social support; social mobility related to health; knowledge, attitudes and values; and behavioural risk factors (Black 1980; Macintyre 1986; Hart 1986; National Health Strategy 1992).

Many risk factors, biomedical and behavioural, are strongly related to socioeconomic status and their unequal distribution in society is believed to contribute to the inverse gradient between socioeconomic status and cardiovascular mortality (Marmot et al. 1984; Pocock et al. 1987; Najman 1994). It follows that monitoring socioeconomic differentials in cardiovascular risk factors is important for understanding trends in the socioeconomic distribution of cardiovascular disease, and to support the development of cost-effective and equitable strategies for its prevention and treatment.

In recent years, a wide variety of initiatives have been undertaken to improve the risk factor profile of Australians and chapter 6 demonstrated favourable trends in the prevalence of cigarette smoking, some dietary behaviours and participation in lighter exercise among adults. Mean blood pressure declined significantly but changes in blood lipids were small. Body fatness increased markedly during the past decade, especially in women. What is not known is whether socioeconomic groups responded differently in adopting healthier lifestyles and how this affected socioeconomic inequalities in biomedical risk factors. To answer these questions, data from the Risk Factor Prevalence Study were examined for socioeconomic inequalities in behavioural and biomedical risk factor levels and for trends in any such inequalities.

8.2 Methods

Indicator of socioeconomic status

Measures of education, occupation and income either singly or as a composite score, are commonly used as indicators of socioeconomic status. These indicators are invariably correlated but are believed to measure different aspects of socioeconomic circumstances (Winkleby et al. 1992; Liberatos et al. 1988). Only educational attainment was collected in a consistent manner across all three surveys and it has therefore has been used to indicate socioeconomic status in this analysis. Educational attainment is considered to be related to values and attitudes which result in healthier behaviours, greater use of preventive health services, and a more positive social and physical environment (Winkleby et al. 1990). It is usually complete by early adulthood and is available for all men and women regardless of employment status (Winkleby et al. 1990). Education has been found to be strongly associated with cardiovascular mortality (Holme et al. 1980) and with coronary risk factors (Jacobsen and Thelle 1988; Reynes et al. 1993). It has been recommended as the most judicious single measure of socioeconomic status for use in epidemiological studies (Winkleby et al. 1992).

Educational attainment was determined by the response to the request `please indicate the highest level of education you have completed'. Education was categorised into groups labelled low (no schooling, primary school or some high school), medium (completed high school at Year 12 or equivalent), and high attainment (completed tertiary qualification at university, college of advanced education or other tertiary institution).

The level of educational attainment of Australians generally has been increasing steadily in recent years (Australian Bureau of Statistics 1994) and this is clearly reflected in the survey samples (Table 8.1). Also clearly shown is that younger survey participants are more likely to have experienced medium (post-compulsory) education than those in the older age group. In addition, high (tertiary) education was reported more often by men than women. Conducting the analysis separately for two broad age groups (25-44, 45-64 years) for men and women allows for an age-sex specific effect, and recognises that post-compulsory education may have a different meaning for different age groups, and for men and women. The trend towards increasing homogeneity of educational attainment may affect the future usefulness of educational attainment as a marker of socioeconomic status.

TABLE 8.1 *Sample distribution by educational attainment, sex, age group and year*

	Men 25-44			Men 45-64			Women 25-44			Women 45-64		
Education	1980	1983	1989	1980	1983	1989	1980	1983	1989	1980	1983	1989
						Sample numbers						
Low	454	555	524	788	911	697	633	734	648	892	1014	847
Medium	532	742	502	321	441	311	512	850	523	338	564	317
High	451	739	621	219	352	330	317	521	569	146	191	208
Total	1437	2036	1647	1328	1704	1338	1462	2105	1740	1376	1769	1372
						Per cent						
Low	31.6	27.3	31.8	59.3	53.5	52.1	47.3	34.9	37.2	64.8	57.3	61.7
Medium	37.0	36.4	30.5	24.2	25.9	23.2	38.3	40.4	30.1	24.6	31.9	23.1
High	31.4	36.3	37.7	16.5	20.7	24.7	23.7	24.8	32.7	10.6	10.8	15.2
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

Statistical analysis

For continuous variables (eg blood pressure), associations with educational attainment over time were examined by analysis of covariance. The model included survey year, age, survey centre and birthplace (Australian-born or overseas-born) as independent variables to allow for differences in the demographic composition of participants over time. An interaction term for educational attainment and year of survey was included to allow for different trends in different education levels. Age was included as a covariate because mean age varied between educational attainment groups even within the broad age strata considered. Use of oral contraceptives was introduced as a factor when analysing data on blood lipids for women aged 25-44.

Multiple logistic regression was used to examine changes in categorical variables over time (eg proportion of smokers) using the same independent

variables as for continuous variables. Using high (tertiary) education in 1980 as the reference group, prevalence odds ratios were derived from the maximum likelihood parameter estimates of the model. Odds ratios for selected risk factors were graphically displayed using a $\log_2$ scale to enable comparison of time trends above and below unity.

The tables which follow show age-sex specific risk factor levels at each survey time, for each category of educational attainment. Also shown are differences between high and low educational attainment (inequalities) for each survey year, and time trends for each level of educational attainment, all adjusted for age and survey design parameters. Trends and inequalities are represented graphically for selected risk factors at the end of the chapter (Figures 7.1 to 7.3).

8.3 Trends in risk factor inequalities

Blood pressure

Generally, falls in blood pressure occurred at each level of educational attainment, with consequent reductions in the prevalence of hypertension (Table 8.2, Figure 8.1).

An educational gradient tended to favour those with higher education, however, the gradient was not statistically significant at all points in time. Some recent studies have reported a negative association between blood pressure and level of education in women but not in men (Garrison et al. 1993; Winkleby et al. 1992; Helmert et al. 1990). Others have found the relationship in both men and women (Jacobsen and Thelle 1988), and others have found no such relationship (Helmert et al. 1990). This may reflect the dynamic influence of other related factors such as diet, treatment for hypertension and smoking behaviour (Jacobsen and Thelle 1988). The results of this study demonstrate that the association can fluctuate over time within the same population subgroup.

The cause(s) of the falls in mean blood pressure over time remains unknown. The sizes of the decreases, which occurred for each level of educational attainment in each age-sex group, are too great to be explained by measurement error (chapter 5). Blood pressure was highly correlated with body mass index and to a lesser extent with smoking status. Controlling for these variables did not explain the falls; in fact many of the decreases were magnified. The estimates of trends and inequalities were essentially unaffected by excluding from the analysis subjects on anti-hypertensive medication.

TABLE 8.2 *Blood pressure by educational attainment, sex, age group and year*

	Men 25-44			Men 45-64			Women 25-44			Women 45-64		
Education	1980	1983	1989	1980	1983	1989	1980	1983	1989	1980	1983	1989
Systolic blood pressure (mmHg)												
Low	129.2	128.8	124.9	140.7	143.2	137.7	118.8	118.0	116.8	139.0	138.8	134.1
Medium	128.8	127.7	124.6	141.3	140.6	135.2	117.2	117.1	114.9	135.6	134.9	133.9
High	125.0	124.8	123.7	137.7	133.0	134.3	115.5	114.7	113.9	134.9	130.8	128.4
Inequalities(a)	*3.6⁺*	*3.6⁺*	*1.0*	*1.9*	*8.4⁺*	*1.5*	*2.2**	*2.1#*	*1.9**	*2.5*	*7.2⁺*	*3.3**
Diastolic blood pressure (mmHg)												
Low	84.4	82.3	80.7	88.4	87.6	85.4	76.7	75.5	75.1	85.9	83.8	81.7
Medium	83.1	80.5	79.8	89.2	86.6	85.4	74.4	74.9	73.3	85.1	81.4	81.3
High	81.3	79.0	80.0	88.3	82.9	85.2	73.8	72.8	72.7	83.7	80.4	80.6
Inequalities(a)	*1.4**	*2.1⁺*	*-0.0*	*0.5*	*4.0⁺*	*0.3*	*1.3**	*1.2**	*1.2**	*1.3*	*2.7⁺*	*0.2*
Hypertension (%)												
Low	18.5	14.2	9.7	38.5	41.6	34.3	8.1	7.0	5.6	39.5	35.4	29.4
Medium	16.5	11.5	8.6	39.6	35.6	30.2	7.2	5.1	3.4	32.8	27.0	27.1
High	11.3	7.0	9.5	38.8	24.2	22.4	4.4	3.1	1.9	29.5	24.6	15.9
Inequalities(a)	*1.43*	*1.82#*	*0.95*	*0.89*	*2.00⁺*	*1.65#*	*1.47*	*1.84**	*2.42**	*1.36*	*1.55**	*1.82#*
Trends (b)												
Variable	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
SBP (mmHg)	*-4.4⁺*	*-4.6⁺*	*-1.8**	*-3.9⁺*	*-6.1⁺*	*-3.5**	*-2.9⁺*	*-2.7⁺*	*-2.6#*	*-4.9⁺*	*-2.8*	*-5.7#*
DBP (mmHg)	*-3.5⁺*	*-3.7⁺*	*-2.1⁺*	*-3.0⁺*	*-3.8⁺*	*-3.2⁺*	*-2.0⁺*	*-1.5**	*-2.0#*	*-4.1⁺*	*-4.1⁺*	*-3.0#*
Hypertension	*0.48⁺*	*0.43⁺*	*0.72*	*0.81*	*0.65**	*0.44⁺*	*0.61**	*0.43#*	*0.37**	*0.64⁺*	*0.71*	*0.48#*

(a) Differences in level (low-high educational attainment) or odds ratios (low/high educational attainment) by survey year adjusted for age and survey design parameters.
(b) Differences in level (1989-1980) or odds ratios (1989/1980) by educational attainment adjusted for age and survey design parameters. * $p<0.05$, # $p<0.01$, + $p<0.001$.

Blood lipids

Educational gradients and time trends for total cholesterol and raised cholesterol were not pronounced (Table 8.3, Figure 8.1), however, the results do suggest an inverse association between average total cholesterol and level of education among women but not among men, a finding reported elsewhere (Winkleby et al. 1992). However, the general body of literature reveals inconsistent results both in terms of direction and strength of the association (Reynes et al. 1993; Helmert et al. 1990; Jacobsen and Thelle 1988), which may be related to variation in the level of public concern and individual awareness in the communities in which the studies were conducted.

Educational attainment and high-density lipoprotein cholesterol were more strongly associated in women than men (Table 8.3), a pattern repeated for low-density lipoprotein cholesterol (Table 8.4). Women exhibited educational gradients in TC/HDL which favoured high education and there were no statistically significant trends over time (Table 8.4). Low-density lipoprotein cholesterol levels decreased significantly among older women with low or medium education.

TABLE 8.3 ***Total cholesterol, raised cholesterol and high-density lipoprotein by educational attainment, sex, age group and year***

	Men 25-44			Men 45-64			Women 25-44			Women 45-64		
Education	1980	1983	1989	1980	1983	1989	1980	1983	1989	1980	1983	1989
						Total cholesterol (mmol/L)						
Low	5.52	5.49	5.55	5.90	5.96	5.92	5.27	5.23	5.25	6.27	6.17	6.07
Medium	5.55	5.42	5.45	6.06	5.95	5.97	5.14	5.21	5.17	6.12	6.11	6.06
High	5.43	5.42	5.38	5.95	5.87	5.86	5.04	5.10	5.11	5.95	6.03	5.73
Inequalities(a)	*-.02*	*-.02*	*.15**	*-.07*	*.04*	*.04*	*.19#*	*.09*	*.09*	*.18*	*.05*	*.14*
						Raised cholesterol (%)						
Low	15.3	14.5	17.1	24.8	28.2	25.9	9.1	9.8	9.8	37.4	34.2	34.0
Medium	16.5	14.1	17.6	32.1	28.4	30.1	8.0	9.3	10.4	34.5	31.5	32.9
High	16.5	13.4	13.6	26.1	26.1	28.5	6.5	6.4	7.7	32.4	29.1	24.1
Inequalities(a)	*0.75*	*0.95*	*1.30*	*0.89*	*1.02*	*0.82*	*1.45*	*1.58*	*1.26*	*1.05*	*1.12*	*1.27*
						High-density lipoprotein (mmol/L)						
Low	1.23	1.22	1.20	1.25	1.25	1.19	1.45	1.48	1.39	1.52	1.54	1.51
Medium	1.24	1.25	1.22	1.25	1.22	1.18	1.48	1.52	1.49	1.52	1.62	1.57
High	1.21	1.26	1.19	1.22	1.26	1.18	1.48	1.58	1.51	1.61	1.63	1.54
Inequalities(a)	*.03*	*-0.4**	*.01*	*.03*	*-.02*	*.01*	*-.04*	*-.10+*	*-.13+*	*-.09**	*-.09#*	*-.03*
						Trends(b)						
Variable	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
TC (mmol/L)	*.06*	*-.13*	*-.11*	*.01*	*-.08*	*-.09*	*-.04*	*.01*	*.07*	*-.19+*	*-.11*	*-.15*
Raised TC	*1.23*	*1.04*	*0.71*	*1.05*	*0.92*	*1.14*	*1.06*	*1.31*	*1.22*	*0.87*	*0.86*	*0.72*
HDL (mmol/L)	*-.04*	*-.02*	*-.02*	*-.06#*	*-.08#*	*-.04*	*-.06#*	*.01*	*.03*	*-.01*	*.05*	*-.07*

(a) Differences in level (low-high educational attainment) or odds ratios (low/high educational attainment) by survey year adjusted for age and survey design parameters.
(b) Differences in level (1989-1980) or odds ratios (1989/1980) by educational attainment adjusted for age and survey design parameters. * $p<0.05$, # $p<0.01$, + $p<0.001$.

The overall lipid picture in this study is one which favours women of high educational attainment who, in addition to lower total cholesterol levels, also had higher average levels of high-density lipoprotein cholesterol, lower triglyceride, lower low-density lipoprotein cholesterol and a lower TC/HDL ratio.

The dietary messages of the 1980s, to reduce the intake of saturated fat, generally had little effect on the lipid profile of any population subgroup. Younger women experienced no change in average total cholesterol at any level of educational attainment. Average total cholesterol levels decreased in older women, but only reached statistical significance in those of low educational attainment. Among men, there were few significant changes in the lipid profile of any educational group, the most notable trends being statistically significant increases in TC/HDL ratios among men of low educational attainment.

TABLE 8.4 ***Triglyceride, low-density lipoprotein and total cholesterol/ high-density lipoprotein by educational attainment, sex, age group and year***

	Men 25-44			Men 45-64			Women 25-44			Women 45-64		
Education	1980	1983	1989	1980	1983	1989	1980	1983	1989	1980	1983	1989
						Triglyceride (mmol/L)						
Low	1.47	1.29	1.53	1.60	1.52	1.64	0.93	0.88	1.02	1.29	1.17	1.34
Medium	1.37	1.19	1.40	1.68	1.49	1.72	0.85	0.81	0.94	1.11	1.11	1.27
High	1.28	1.13	1.32	1.48	1.35	1.62	0.82	0.75	0.93	1.00	0.97	1.11
Inequalities(a)	*.09*	*.10*	*.19#*	*.10*	*.14**	*.00*	*.11#*	*.14+*	*.08#*	*.22+*	*.16#*	*.15#*
						Low-density lipoprotein (mmol/L)						
Low	3.64	3.70	3.67	3.91	4.05	4.00	3.40	3.35	3.39	4.18	4.09	3.95
Medium	3.69	3.64	3.61	4.06	4.07	4.01	3.27	3.33	3.24	4.09	3.98	3.91
High	3.63	3.67	3.59	4.04	4.02	3.93	3.17	3.18	3.17	3.86	3.95	3.69
Inequalities(a)	*-.09*	*-.04*	*.07*	*-.13*	*.01*	*.06*	*.19#*	*.14#*	*.18#*	*.20**	*.05*	*.10*
						Total cholesterol / High-density lipoprotein						
Low	4.74	4.86	5.01	5.01	5.12	5.29	3.84	3.72	3.97	4.37	4.30	4.32
Medium	4.70	4.62	4.77	5.12	5.31	5.37	3.63	3.62	3.64	4.27	4.00	4.11
High	4.67	4.56	4.80	5.07	5.02	5.27	3.56	3.37	3.54	3.91	3.90	3.93
Inequalities(a)	*-.08*	*.18*	*.19*	*-.04*	*.09*	*.00*	*.27#*	*.35+*	*.41+*	*.34#*	*.31**	*.24*
						Trends(b)						
Variable	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
TG (mmol/L)	*.09*	*.01*	*-.01*	*.03*	*.03*	*.13*	*.07**	*.09#*	*.11#*	*.06*	*.14#*	*.13*
LDL (mmol/L)	*.06*	*-.09*	*-.10*	*.08*	*-.04*	*-.10*	*-.03*	*-.04*	*.01*	*-.21+*	*-.23#*	*-.12*
TC/HDL	*.31#*	*.03*	*.04*	*.25**	*.25*	*.21*	*.11*	*-.00*	*-.03*	*-.04*	*-.19*	*.06*

(a) Differences in level (low-high educational attainment) by survey year adjusted for age and survey design parameters.
(b) Differences in level (1989-1980) by educational attainment adjusted for age and survey design parameters.
* p<0.05, # p<0.01, + p<0.001.

Height and weight

These data demonstrate, for each survey and age-sex group, the well known association between adult height and socioeconomic status. Younger men of high educational attainment were 2.7 cm taller on average than men of the same age of low educational attainment (Table 8.5). The differential for older

men was 3.3 cm, for younger women 1.9 cm, and for older women 2.0 cm (derived from Table 8.5).

TABLE 8.5 ***Weight for height by educational attainment, sex, age group and year***

	Men 25-44			Men 45-64			Women 25-44			Women 45-64		
Education	1980	1983	1989	1980	1983	1989	1980	1983	1989	1980	1983	1989
						Height (cm)						
Low	173.8	173.7	174.6	171.5	171.8	172.1	161.3	161.6	162.1	159.4	159.4	160.4
Medium	176.3	176.1	176.6	173.5	173.7	174.1	162.1	163.1	162.6	161.0	161.4	161.7
High	176.7	176.7	177.0	175.2	175.5	175.6	164.0	163.4	163.6	162.1	161.6	162.3
Inequalities(a)	*-2.6⁺*	*-2.8⁺*	*-2.6⁺*	*-3.6⁺*	*-3.4⁺*	*-3.1⁺*	*-2.5⁺*	*-1.6⁺*	*-1.4⁺*	*-2.4⁺*	*-2.0⁺*	*-1.5#*
						Weight (kg)						
Low	78.3	76.9	79.2	77.1	78.2	79.6	61.8	63.9	65.5	64.3	66.9	68.8
Medium	78.7	77.8	79.6	79.4	79.9	81.9	60.5	62.1	63.2	64.7	64.3	67.4
High	77.1	76.8	79.2	78.8	79.1	81.2	60.1	61.0	62.7	64.3	63.5	66.6
Inequalities(a)	*0.5*	*-0.3*	*-0.3*	*-1.6*	*-0.7*	*-1.6*	*1.3*	*2.4⁺*	*2.4⁺*	*-0.2*	*3.2⁺*	*2.1**
						Body mass index (kg/m2)						
Low	25.3	25.1	25.6	25.8	26.1	26.5	23.3	24.1	24.6	24.8	26.0	26.4
Medium	24.9	24.8	25.2	26.1	26.1	26.7	22.5	23.0	23.5	24.5	24.3	25.4
High	24.3	24.3	24.9	25.4	25.3	25.9	21.9	22.4	23.0	24.0	23.9	24.9
Inequalities(a)	*0.7#*	*0.7⁺*	*0.6#*	*0.4*	*0.7#*	*0.5*	*1.2⁺*	*1.4⁺*	*1.4⁺*	*0.6*	*1.9⁺*	*1.3⁺*
						Overweight or obese (%)						
Low	52.0	46.3	54.8	57.1	61.3	64.2	22.8	30.3	36.2	40.2	50.0	54.7
Medium	44.6	41.9	49.1	57.4	59.6	67.3	17.2	20.5	27.4	35.2	35.2	44.6
High	36.1	37.1	44.5	51.3	49.4	58.1	11.1	16.8	20.8	34.4	33.2	40.1
Inequalities(a)	*1.67⁺*	*1.35**	*1.51⁺*	*1.27*	*1.56⁺*	*1.23*	*2.19⁺*	*1.92⁺*	*1.99⁺*	*1.21*	*1.91⁺*	*1.66#*
						Obese (%)						
Low	6.8	8.7	11.8	12.8	13.7	16.0	8.3	11.6	13.0	12.0	17.7	20.5
Medium	7.0	7.7	8.8	12.8	12.2	15.2	4.2	5.7	7.1	9.6	8.9	15.5
High	6.4	4.1	7.0	7.9	7.4	10.2	1.1	5.0	6.7	6.9	8.0	12.6
Inequalities(a)	*0.94*	*2.04#*	*1.68**	*1.74*	*1.99#*	*1.63**	*7.26⁺*	*2.32⁺*	*1.95#*	*1.78*	*2.36#*	*1.69**
						Trends(b)						
Variable	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
Height (cm)	*0.4*	*0.1*	*0.4*	*0.8**	*0.8*	*0.3*	*0.8**	*0.4*	*-0.3*	*1.1⁺*	*1.0**	*0.2*
Weight (kg)	*0.6*	*0.3*	*1.5**	*2.5⁺*	*2.7#*	*2.5**	*3.3⁺*	*2.4#*	*2.2#*	*4.4⁺*	*2.3**	*2.1*
BMI (kg/m^2)	*0.3*	*0.0*	*0.3*	*0.6#*	*0.6*	*0.6*	*1.1⁺*	*0.9#*	*1.0#*	*1.4⁺*	*0.6*	*0.8*
O'weight/obese	*1.16*	*1.14*	*1.28*	*1.23*	*1.46**	*1.28*	*1.77⁺*	*1.73#*	*1.95#*	*1.68⁺*	*1.34*	*1.23*
Obese	*1.65*	*1.08*	*0.92*	*1.31*	*1.28*	*1.39*	*1.57**	*1.66*	*5.83#*	*1.76⁺*	*1.56*	*1.85*

(a) Differences in level (low-high educational attainment) or odds ratios (low/high educational attainment) by survey year adjusted for age and survey design parameters.
(b) Differences in level (1989-1980) or odds ratios (1989/1980) by educational attainment adjusted for age and survey design parameters. * p<0.05, # p<0.01, + p<0.001.

Reasons for the association are uncertain but adult height has been suggested as a good indicator of net nutritional status and average health status (Carr-Hill 1988). The association may, therefore, reflect differences in nutrition and health during early childhood, and the statistically significant increases in the average height of men (older) and women of lower educational attainment may reflect improvements in conditions during childhood in these socioeconomic groups. Height is known to be a predictor of all cause mortality and coronary heart disease mortality in middle aged men (Marmot et al. 1984).

The average weight of women increased from between 2.1 and 4.4 kg over the study period depending on age and educational attainment, with greater increases among women of low educational attainment. Older men experienced increases of around 2.5 kg regardless of educational attainment. There were consequent increases in average body mass index and in the prevalence of people classified as overweight or obese. The greatest increases in the odds of being overweight or obese were experienced by younger women independent of educational attainment, and by older women of lower education (Table 8.5, Figure 8.1).

The inverse relationship between body mass index and educational attainment was repeated at each survey. The relationship was strongest for women age 25-44 for whom ratios close to 2 were observed in each survey for the relative odds of being overweight or obese (Table 8.5).The prevalence of women in this age group who were overweight or obese in 1989 was still less than that in the other age-sex groups despite the significant increases which occurred at each level of educational attainment.

Body mass index was inversely related to smoking status (not shown), especially among older men and women and to a lesser extent among younger men, but not amongst younger women. Consequently, smoking status could not explain the higher mean body mass index in those of low educational attainment since smoking was more prevalent in that strata. As expected, when smoking status was added to the model the educational differentials were slightly increased (not shown). It is possible to hypothesise, however, that the decreases in the prevalence of smoking over time could have contributed to the increases in mean body mass index. In fact, controlling for smoking status did slightly moderate the increases in mean body mass index among older men and women, but did not completely explain them. This is consistent with an independent analysis (Boyle et al. 1994).

Dietary behaviour

Regarding dietary behaviour, 'not adding salt to food' became more common at each level of educational attainment (Figure 8.2). This was observed for each age-sex group although the data suggest greater improvements among younger tertiary educated men and women (Table 8.6). Significant gradients existed with educational attainment for each age-sex group and were maintained over time. Education gradients were not as marked for 'not eating the fat on meat', and were statistically significant only for younger men and women in 1989. All time trends were statistically significant with the exception of younger men of low educational attainment and older men of medium educational attainment.

TABLE 8.6 *Dietary behaviour by educational attainment, sex, age group and year*

	Men 25-44			Men 45-64			Women 25-44			Women 45-64		
Education	1980	1983	1989	1980	1983	1989	1980	1983	1989	1980	1983	1989
						Do not add salt to food (%)						
Low	na	23.8	37.8	na	25.7	37.3	na	37.6	53.8	na	39.7	50.9
Medium	na	30.9	48.8	na	27.7	42.4	na	41.9	53.7	na	43.3	53.0
High	na	42.2	67.0	na	35.8	53.9	na	46.3	71.2	na	48.7	62.0
Inequalities(a)	*na*	*0.43⁺*	*0.30⁺*	*na*	*0.61⁺*	*0.50⁺*	*na*	*0.71#*	*0.48⁺*	*na*	*0.70**	*0.64#*
						Do not eat fat on meat (%)						
Low	38.8	na	39.7	41.8	na	50.1	50.6	na	59.6	52.0	na	63.0
Medium	41.4	na	48.8	42.4	na	44.7	46.7	na	61.8	51.2	na	67.2
High	43.2	na	52.2	38.4	na	53.9	47.9	na	68.2	50.0	na	62.0
Inequalities(a)	*0.84*	*na*	*0.62⁺*	*1.12*	*na*	*0.82*	*1.13*	*na*	*0.70#*	*1.00*	*na*	*0.98*
						Trends(b)						
Variable	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
Do not add salt	*1.97⁺*	*2.17⁺*	*2.78⁺*	*1.73⁺*	*1.91⁺*	*2.13⁺*	*1.87⁺*	*1.56⁺*	*2.79⁺*	*1.57⁺*	*1.43**	*1.70#*
Do not eat fat	*1.06*	*1.38**	*1.44#*	*1.37#*	*1.05*	*1.87⁺*	*1.46⁺*	*1.87⁺*	*2.36⁺*	*1.60⁺*	*1.94⁺*	*1.65**

na not available, question not asked.
(a) Odds ratios (low/high educational attainment) by survey year adjusted for age and survey design parameters.
(b) Odds ratios (1989/1983) or (1989/1980) by educational attainment adjusted for age and survey design parameters.
* p<0.05, # p<0.01, + p<0.001.

The questions on discretionary use of salt and propensity to eat the fat on meat are useful for monitoring behaviour and awareness of health messages. They are not intended as measures of sodium or saturated fat intake. The advice to avoid salt was adopted across the spectrum of educational attainment but with no suggestion that the educational gradient, which favoured the more highly educated, was diminishing. The results suggest the emergence of educational differentials among younger men and women in respect of attitudes towards eating the fat on meat.

Smoking

In both younger and older men, the prevalence of smoking was consistently less among the higher educated and all groups experienced declines in smoking prevalence (Table 8.7). This suggests that men of all educational levels responded positively to the anti-smoking messages of the 1980s while maintaining the relative differences between groups. The picture is different among both younger and older women, for whom the decline has been less (and not statistically significant) among those of low educational attainment (Table 8.7, Figure 8.2). That is, women of low educational attainment have been least likely to respond to past primary prevention activities and may be an appropriate target group for future health promotion programs. In contrast to the other age-sex groups, there was no clear evidence of an educational gradient for smoking prevalence among older women (Figure 8.2).

TABLE 8.7 *Smoking and alcohol consumption by educational attainment, sex, age group and year*

	Men 25-44			Men 45-64			Women 25-44			Women 45-64		
Education	1980	1983	1989	1980	1983	1989	1980	1983	1989	1980	1983	1989
						Current smokers (%)						
Low	48.9	46.0	34.2	40.5	36.3	28.3	31.5	28.1	29.1	22.6	23.7	19.5
Medium	40.0	40.2	34.3	34.1	38.0	25.1	31.4	28.5	25.6	29.0	21.8	16.7
High	26.4	24.0	18.2	24.3	20.2	13.4	20.5	18.0	13.5	26.0	22.5	15.4
Inequalities(a)	*2.75⁺*	*2.78⁺*	*2.38⁺*	*2.12⁺*	*2.29⁺*	*2.56⁺*	*1.97⁺*	*1.97⁺*	*2.80⁺*	*0.88*	*1.09*	*1.40*
						Light alcohol consumption (%)						
Low	74.9	75.1	80.8	70.7	68.8	73.2	75.7	68.9	71.7	65.6	55.4	57.3
Medium	84.8	80.9	85.9	75.4	75.5	76.8	85.4	78.9	75.1	75.7	70.0	74.4
High	88.5	87.8	88.1	81.7	81.3	77.9	84.5	83.3	81.9	67.8	73.8	76.4
Inequalities(a)	*0.41⁺*	*0.45⁺*	*0.60#*	*0.56#*	*0.53⁺*	*0.78*	*0.61#*	*0.48⁺*	*0.58⁺*	*1.00*	*0.47⁺*	*0.45⁺*
						Moderate / heavy alcohol consumption (%)						
Low	16.1	10.6	7.9	16.4	13.9	8.9	4.6	4.1	2.3	5.5	6.1	4.9
Medium	10.3	11.3	5.8	15.6	12.0	10.3	4.7	4.5	3.3	6.8	8.0	5.7
High	5.8	5.5	3.1	9.6	5.4	7.3	6.0	5.8	3.3	13.0	4.7	3.8
Inequalities(a)	*3.28⁺*	*2.03⁺*	*2.67⁺*	*1.87**	*2.94⁺*	*1.34*	*0.78*	*0.69*	*0.72*	*0.43#*	*1.47*	*1.45*
						Trends(b)						
Variable	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
Current smokers	*0.55⁺*	*0.81*	*0.64#*	*0.58⁺*	*0.64**	*0.48#*	*0.89*	*0.76*	*0.63**	*0.83*	*0.51⁺*	*0.52**
Light alcohol	*1.45**	*1.12*	*1.00*	*1.09*	*1.03*	*0.78*	*0.79*	*0.51⁺*	*0.83*	*0.69⁺*	*0.97*	*1.53*
Mod/hvy alcohol	*0.42⁺*	*0.52#*	*0.52**	*0.55⁺*	*0.70*	*0.76*	*0.48**	*0.69*	*0.52*	*0.89*	*0.88*	*0.27#*

(a) Odds ratios (low/high educational attainment) by survey year adjusted for age and survey design parameters.
(b) Odds ratios (1989/1980) by educational attainment adjusted for age and survey design parameters.
* $p<0.05$, # $p<0.01$, ⁺ $p<0.001$.

Alcohol consumption

In 1980, the prevalence of moderate or heavy drinkers was highest among both younger and older men of low educational attainment (Table 8.7), and the significant reduction over the 1980s is a positive finding. Among younger men, the prevalence of moderate or heavy drinking declined at each level of educational attainment, although the educational gradient persisted. The data suggest a negative association between educational attainment and moderate or heavy alcohol consumption in men, but a positive association in women, a finding consistent with other studies (Garrison et al. 1993; Woodward et al. 1992). The high prevalence of moderate or heavy drinking reported in 1980 by older women with high educational attainment had significantly declined by 1983 and was maintained in 1989 (Table 8.7). The sharp decrease is, perhaps, a good example that such associations are not immutable. The surveillance of light alcohol consumption is relevant to coronary risk given recent evidence that modest alcohol consumption is linked to lower cardiovascular risk (Marmot and Brunner 1991; Jackson et al. 1991). In this study the educational gradient invariably favours those of higher educational attainment.

Exercise

The lack of standardisation in the definition and assessment of physical activity in epidemiological research in Australia (Lee 1993) means that comparisons between surveys are uncommon. This present study used the same self-reported 2-week recall questionnaire technique to measure leisure-time activity in two surveys six years apart which means that, although comparison of absolute levels of exercise with other studies may be problematic, the data are suitable for the internal identification of exercise differentials, both socioeconomic and secular.

All forms of leisure-time exercise were strongly associated with educational attainment (Table 8.8). Those of high educational attainment were more likely to participate in exercise than those of low attainment (Figure 8.2). There was little change over time for men in participation rates for aerobic exercise or vigorous exercise. For women, the data suggest changes in participation rates for aerobic exercise for those of medium educational attainment, increasing participation for younger women and decreasing for older women. There appears to have been a general increase in walking as a form of exercise regardless of level of education, however, the data are self-reported and there is no way of telling whether the trend reflects increased awareness or actuality. Using 'no exercise in the past 2 weeks' as a summary measure shows that the

educational gradient observed for physical inactivity during leisure time persisted over time, with the greatest improvement shown by older men of low educational attainment.

TABLE 8.8 ***Exercise during leisure time by educational attainment, sex, age group and year***

	Men 25-44			Men 45-64			Women 25-44			Women 45-64		
Education	1980	1983	1989	1980	1983	1989	1980	1983	1989	1980	1983	1989
						Aerobic exercise (%)						
Low	na	7.2	7.6	na	3.9	4.5	na	4.9	3.9	na	1.7	1.8
Medium	na	12.8	12.9	na	6.4	4.8	na	5.9	9.4	na	4.8	1.9
High	na	15.3	12.3	na	10.1	12.7	na	10.2	9.5	na	7.5	5.8
Inequalities(a)	*na*	*0.45⁺*	*0.57#*	*na*	*0.43⁺*	*0.39⁺*	*na*	*0.51#*	*0.41⁺*	*na*	*0.22⁺*	*0.34#*
						Vigorous exercise (%)						
Low	na	29.0	31.7	na	10.8	12.8	na	25.0	27.5	na	9.7	12.9
Medium	na	43.8	43.2	na	19.5	18.3	na	35.6	32.3	na	17.2	15.8
High	na	53.8	52.2	na	31.6	29.7	na	48.3	41.2	na	19.8	26.9
Inequalities(a)	*na*	*0.39⁺*	*0.40⁺*	*na*	*0.33⁺*	*0.43⁺*	*na*	*0.40⁺*	*0.59⁺*	*na*	*0.49⁺*	*0.49⁺*
						Walking (%)						
Low	na	36.9	46.4	na	43.4	52.3	na	47.0	53.0	na	45.6	51.6
Medium	na	44.7	49.0	na	53.3	55.6	na	57.4	59.3	na	59.9	65.0
High	na	52.8	58.8	na	60.8	66.1	na	65.6	73.8	na	62.8	68.8
Inequalities(a)	*na*	*0.54⁺*	*0.60⁺*	*na*	*0.46⁺*	*0.54⁺*	*na*	*0.51⁺*	*0.42⁺*	*na*	*0.50⁺*	*0.47⁺*
						No exercise (%)						
Low	na	42.9	35.1	na	48.0	38.5	na	36.6	32.7	na	44.6	39.8
Medium	na	27.4	28.5	na	31.3	30.2	na	23.2	27.0	na	31.3	29.0
High	na	17.2	15.8	na	20.5	19.1	na	15.9	15.5	na	24.6	20.7
Inequalities(a)	*na*	*3.31⁺*	*3.03⁺*	*na*	*3.56⁺*	*2.60⁺*	*na*	*2.70⁺*	*2.46⁺*	*na*	*2.39⁺*	*2.46⁺*
						Trends(b)						
Variable	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
Aerobic exercise	*1.00*	*1.04*	*0.79*	*1.25*	*0.79*	*1.40*	*0.79*	*1.69**	*0.97*	*1.07*	*0.39**	*0.71*
Vig. exercise	*1.02*	*1.00*	*1.00*	*1.23*	*1.01*	*0.96*	*1.15*	*0.89*	*0.78**	*1.37**	*0.97*	*1.38*
Walking	*1.38#*	*1.14*	*1.24**	*1.43⁺*	*1.13*	*1.23*	*1.22*	*1.09*	*1.48#*	*1.25**	*1.21*	*1.31*
No exercise	*0.80*	*1.06*	*0.87*	*0.67⁺*	*0.89*	*0.92*	*0.85*	*1.19*	*0.93*	*0.83**	*0.89*	*0.80*

na not available, question not asked.

(a) Odds ratios (low/high educational attainment) by survey year adjusted for age and survey design parameters.

(b) Odds ratios (1989/1983) by educational attainment adjusted for age and survey design parameters.

* $p<0.05$, # $p<0.01$, ⁺ $p<0.001$.

The strong positive association between educational attainment and physical activity during leisure time, in all subgroups and both surveys, is supported by similar findings from other studies (Garrison et al. 1993; Helmert et al. 1990; Jacobsen and Thelle 1988). The implications of this for coronary risk among

women is clear, but not so for men. This is because other studies have found, among men but not women, that the lower levels of participation in physical activity during leisure time among those of lower educational attainment may be compensated for by increased physical activity at work (Garrison et al. 1993; Woodward et al. 1992). This could well apply in the present study, as the correlation between educational attainment and occupational status for men was highly significant ($r=+0.54$; $p<.0001$); that is, men of lower educational attainment were more likely to be in manual occupations. Further, it is known that leisure time activity and exercise at work are both associated with reduced coronary risk, although their relative importance is unclear (Salonen et al. 1988).

Multiple risk factors

The prevalence of multiple *behavioural* risk factors displayed strong (inverse) associations with educational attainment for each age-sex group, resulting in prevalence odds ratios around 2 to 5 for low compared to high educational attainment (Table 8,9). That is, adults with lower education remained more likely to report an unhealthy lifestyle. Nevertheless, all education groups responded positively to the health education messages of the eighties and adopted healthier profiles of risk factor behaviours. Certain combinations of behaviours were more common than others (not shown). Cigarette smoking and excess drinking were correlated at each level of educational attainment but especially among older women of high educational attainment. Among men, cigarette smoking was also correlated with lack of exercise and with higher discretionary use of salt.

An inverse association with educational attainment was also consistently observed for the prevalence of multiple *biomedical* risk factors among both younger and older women. The results suggest a worsening of the biomedical risk factor profile for younger women irrespective of educational attainment, but some improvement for older women, although the trends were statistically significant only for those of medium education. There were no statistically significant trends for men.

The simultaneous occurrence of two or more of the three *classical* risk factors for coronary heart disease was invariably associated with educational attainment, with those of high education least likely to have multiple risk factors. However, only among young men were the educational gradients statistically significant at each survey. Generally, statistically significant decreases in the prevalence of multiple risk factors were observed at each education level, except for young women where prevalences were already less than 6%. This pattern was

observed also for the average number of classical risk factors per person (Figure 8.3) and is generally consistent with parallel declines in mortality.

TABLE 8.9 *Multiple risk factors by educational attainment, sex, age group and year*

	Men 25-44			Men 45-64			Women 25-44			Women 45-64		
Education	1980	1983	1989	1980	1983	1989	1980	1983	1989	1980	1983	1989
2-3 classical(a) risk factors(%)												
Low	14.9	11.4	10.0	19.0	20.3	14.5	5.4	4.1	3.6	18.2	14.7	11.4
Medium	12.3	8.7	9.3	23.8	20.4	14.5	3.3	4.4	3.8	19.0	12.3	9.5
High	7.8	5.3	4.5	17.9	8.3	10.7	2.5	1.3	2.6	15.1	8.4	7.7
Inequalities(b)	*1.80#*	*2.06+*	*2.35+*	*1.00*	*2.57+*	*1.32*	*1.95*	*2.87**	*1.20*	*1.10*	*1.69*	*1.26*
2-4 biomedical(c) risk factors (%)												
Low	9.8	10.6	11.5	18.6	19.5	21.7	7.1	9.4	10.6	21.9	20.0	19.4
Medium	7.3	8.4	9.9	22.9	17.3	20.5	2.2	4.8	6.1	20.8	12.1	14.5
High	9.4	5.2	8.0	18.4	11.7	14.3	1.4	2.8	4.8	13.2	10.3	9.3
Inequalities(b)	*0.85*	*1.86#*	*1.40*	*0.98*	*1.74#*	*1.58**	*4.14#*	*3.03+*	*2.11#*	*1.59*	*2.01#*	*1.96#*
2-4 behavioural(d) risk factors (%)												
Low	na	43.4	29.1	na	44.0	30.6	na	25.6	19.5	na	27.5	20.3
Medium	na	33.8	21.9	na	35.7	21.2	na	20.1	15.6	na	20.2	12.9
High	na	15.2	7.2	na	18.2	11.5	na	12.3	6.9	na	18.3	7.7
Inequalities(b)	*na*	*4.08+*	*5.21+*	*na*	*3.48+*	*3.44+*	*na*	*2.48+*	*3.31+*	*na*	*1.75#*	*3.26+*
Trends(e)												
Variable	Low	Med	High	Low	Med	High	Low	Med	High	Low	Med	High
Classical(a)	*0.67**	*0.71*	*0.52**	*0.71**	*0.54#*	*0.54**	*0.62*	*1.14*	*1.00*	*0.59+*	*0.43+*	*0.51*
Biomedical(c)	*1.21*	*1.30*	*0.73*	*1.17*	*0.85*	*0.72*	*1.56**	*3.04#*	*3.06**	*0.84*	*0.58**	*0.68*
Behavioural(d)	*0.55+*	*0.55+*	*0.43+*	*0.58+*	*0.49+*	*0.59**	*0.70#*	*0.73**	*0.52#*	*0.67+*	*0.59#*	*0.36#*

na not available, some questions not asked.
(a) high diastolic blood pressure, high total cholesterol, regular cigarette smoking.
(b) Odds ratios (low/high educational attainment) by survey year adjusted for age and survey design parameters.
(c) high diastolic blood pressure, high total cholesterol, low high-density lipoprotein cholesterol, obesity.
(d) regular cigarette smoking, no leisure time physical activity, moderate to heavy alcohol intake, almost always add salt to food.
(e) Odds ratios (1989/1980) or (1989/1983) by educational attainment adjusted for age and survey design parameters.
* p<0.05, # p<0.01, + p<0.001.

Overall, the data suggest lower cardiovascular risk among the tertiary educated. Decreased risk of coronary events with increasing educational attainment is consistent with the known socioeconomic gradients in mortality from coronary heart disease and stroke. Overall, the results support the view that differentials in cardiovascular mortality may, at least in part, be ascribed to differences in biomedical and behavioural risk factors.

A supplementary analysis of the combined use of oral contraception and cigarettes revealed an increase in prevalence from 8% to 17% between 1980 and

1989 among women aged 25-34 of low educational attainment (p=.01) and a marked educational gradient appeared in 1989 (p<.001). Conjoint use among women aged 35-44, who are more at risk of hypertension, was low at around 2% in 1989 (Table 8.10).

TABLE 8.10 ***Oral contraceptive use and smoking rates in women, by educational attainment, age group and year***

	Taking o/c pill (%)			Smoking (%)			Both behaviours (%)		
Education	1980	1983	1989	1980	1983	1989	1980	1983	1989
				25-34 years					
Low	24.1	29.3	43.2	37.1	31.0	39.0	8.3	11.4	17.4
Medium	32.9	35.0	37.7	29.3	30.2	27.2	13.3	11.1	13.0
High	29.9	37.7	34.5	20.4	18.0	13.2	6.0	9.3	5.2
				35-44 years					
Low	11.2	12.7	11.8	20.7	18.1	13.8	3.4	1.8	1.8
Medium	9.3	10.9	14.3	34.3	26.3	23.8	3.3	3.2	0.8
High	9.5	9.5	9.2	26.7	26.3	23.4	3.0	3.1	2.5

Studies in the 1970s showed that oral contraceptive use increased the risk of cardiovascular events such as heart attack and stroke and that this risk was greatly enhanced in the presence of smoking. The sharp increase in the prevalence of conjoint use among younger women of low educational attainment would be of concern were it not for the fact that the formulation of oral contraceptives has changed markedly over the past 20 years and it is likely that the risk associated with modern low-dose pills is lower than that reported more than a decade previously (Milne and Vessey 1992).

8.4 Discussion

The Risk Factor Prevalence Study provides a unique opportunity to examine trends in socioeconomic differentials in cardiovascular risk factors in a representative sample of urban Australians. Few studies have reported trends in risk factor inequalities and those that have tend to be of single local or regional communities (Winkleby et al. 1990; Brannstrom et al. 1993; Luepker et al. 1993). The present data analysis is based on nation-wide probability sample surveys of urban living Australian adults aged 25-64 with an average sample size of 6,400 respondents. The results improve our understanding of cardiovascular disease aetiology in society, and help in formulating policy and determining health promotion activities.

The effect of differential response bias by educational attainment on the associations observed will have been to produce underestimates of the true risk factor gradients by educational attainment. The argument is equivalent to that

used in chapter 7 for occupational differentials. People with an adverse risk factor profile are less likely to participate in surveys such as these (Bergstrand et al. 1983; Criqui et al. 1978). Since risk factors are more prevalent in low socioeconomic groups, it follows that inequalities are likely to be underestimates of the true differentials. Overestimation of the association between educational attainment and risk factors would occur only if propensity to respond was associated with a better risk factor profile among the better educated and a worse profile among the less educated, a phenomenon which has not been reported in other studies. The remarkable consistency in overall response rates over time (75% in each survey) offers some assurance against biased trend estimates, since characteristics of nonrespondents have been found to be relatively stable over time (Sprafka et al. 1990).

It is clear that Australians of different educational attainment have very different risk factor profiles. Socioeconomic inequalities are more pronounced for behavioural than biomedical risk factors, and for hypertension and body fatness than blood lipids. For both biomedical and behavioural risk factors, the strength of the associations varies with sex and age and inequalities are more common among women than men. However, the cross-sectional nature of the data imposes limitations on the interpretation of these associations. They do not necessarily imply that increasing the general level of educational attainment in society will result in improved levels of risk factors, although this is plausible.

The lower socioeconomic group has improved its risk factor profile but its relative disadvantage compared with the higher socioeconomic group persists. This suggests that the health promotion activities in Australia have been effective in reaching the lower socioeconomic groups but that the challenge to reduce inequalities remains. The increase in weight is an unfavourable development shared by each socioeconomic group which, coupled with little reduction in smoking prevalence among women of low socioeconomic standing, might be expected to retard cardiovascular benefits in that population subgroup unless they are addressed.

Educational attainment may be a surrogate for other factors (eg income which makes it easier to make healthy lifestyle choices) but it is also possible that exposure to higher formal education has a direct effect on risk factor levels and behaviours. The effect might be through the curriculum content or the intellectual training involved. Both might increase peoples ability to understand and relate to health promotion exposure after formal education has

ceased (Jacobsen and Thelle 1988) and improve health literacy. Regardless of the mechanism behind the association between education and risk factors, it is plausible that the steady increase in educational attainment in Australia has been an important factor in the general improvement in the nation's risk factor profile and in the decrease in mortality from coronary heart disease.

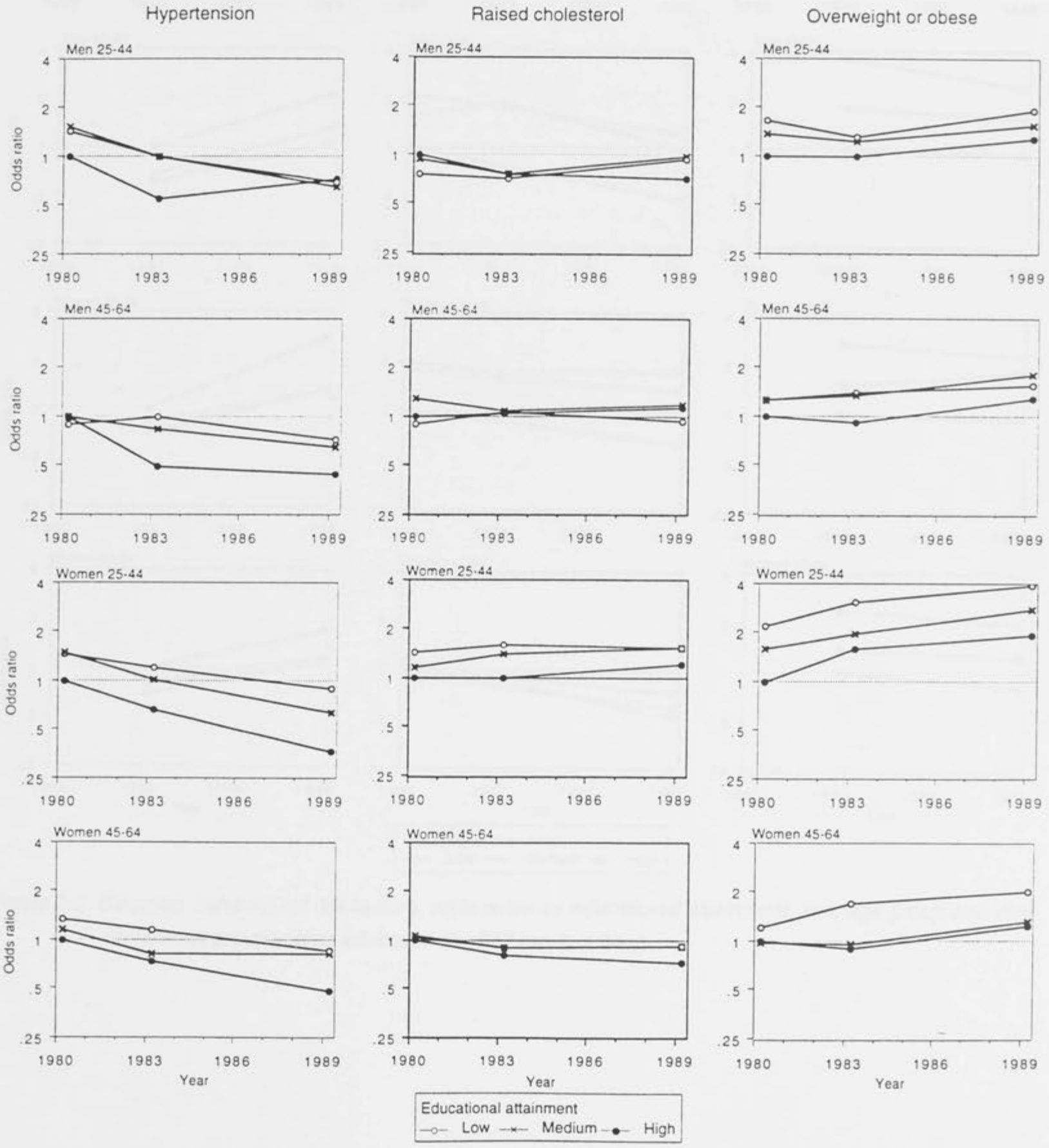

Figure 8.1 *Selected biomedical risk factors, odds ratios by educational attainment, sex, age group and year. Reference group is high education in 1980 (odds ratio=1)*

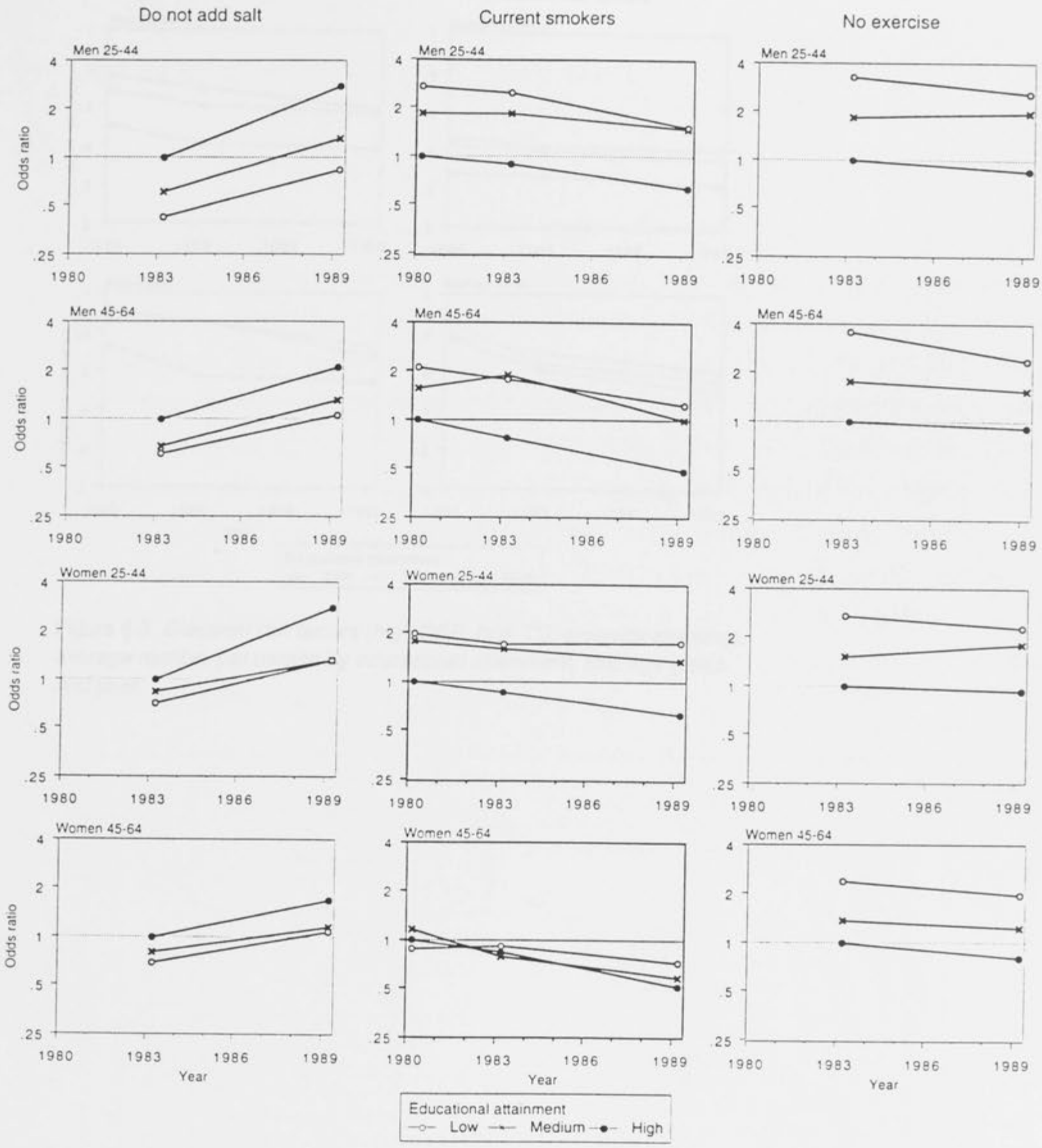

Figure 8.2 *Selected behavioural risk factors, odds ratios by educational attainment, sex, age group and year. Reference group is high education in 1980 (odds ratio=1)*

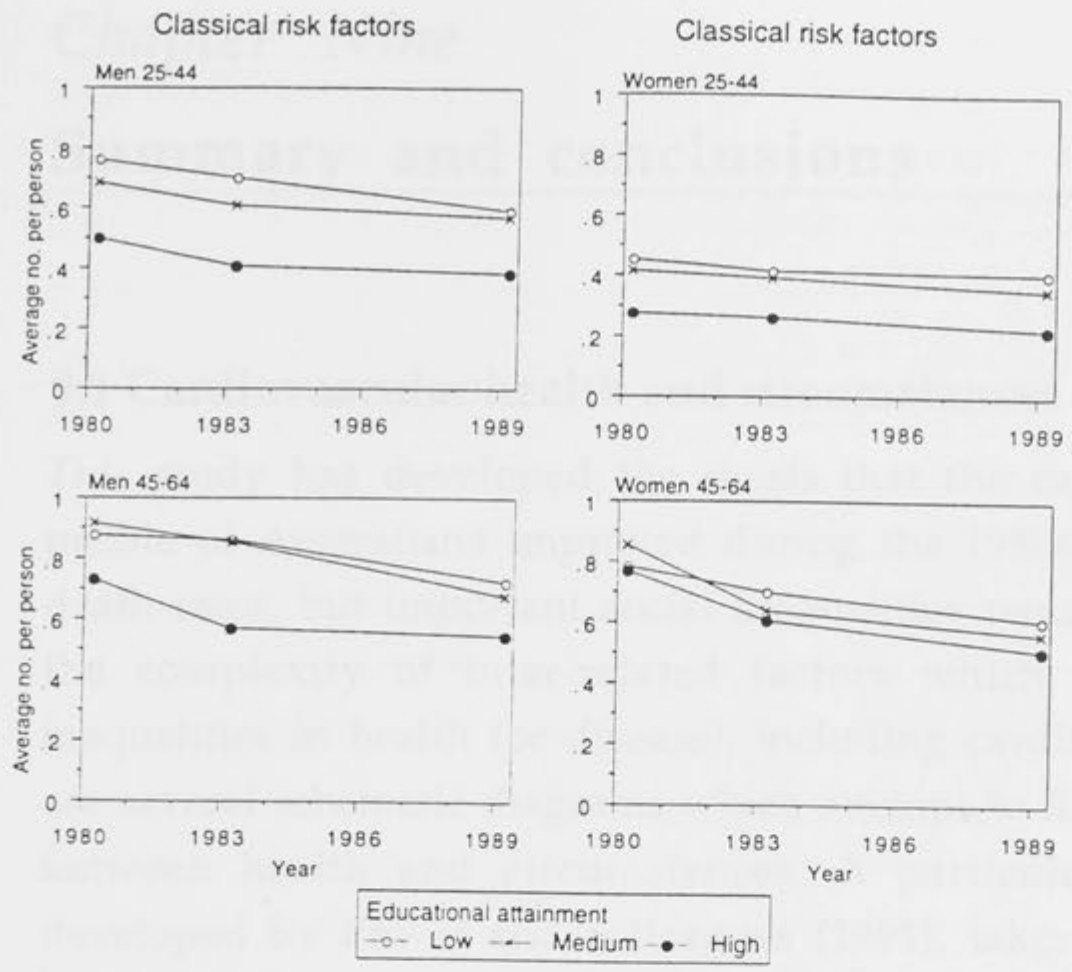

Figure 8.3 *Classical risk factors (high DBP, high TC, cigarette smoking), average number per person by educational attainment, sex, age group and year.*

Chapter Nine

Summary and conclusions

9.1 Cardiovascular health and circumstances

This study has developed the thesis that the cardiovascular risk factor profile of Australians improved during the 1980s, consistent with falling death rates, but important social inequalities remain. Chapter 2 discussed the complexity of inter-related factors which lie behind trends and inequalities in health (or disease), including cardiovascular disease. There are several schematic diagrams which attempt to illustrate the relationships between health and circumstances. A particularly useful framework, developed by Power and colleagues (1991), takes into account pathways between generations. Their diagram has been modified (see Figure 9.1) to focus more on cardiovascular disease and to provide a summary of the causal pathways, described in chapter 2.

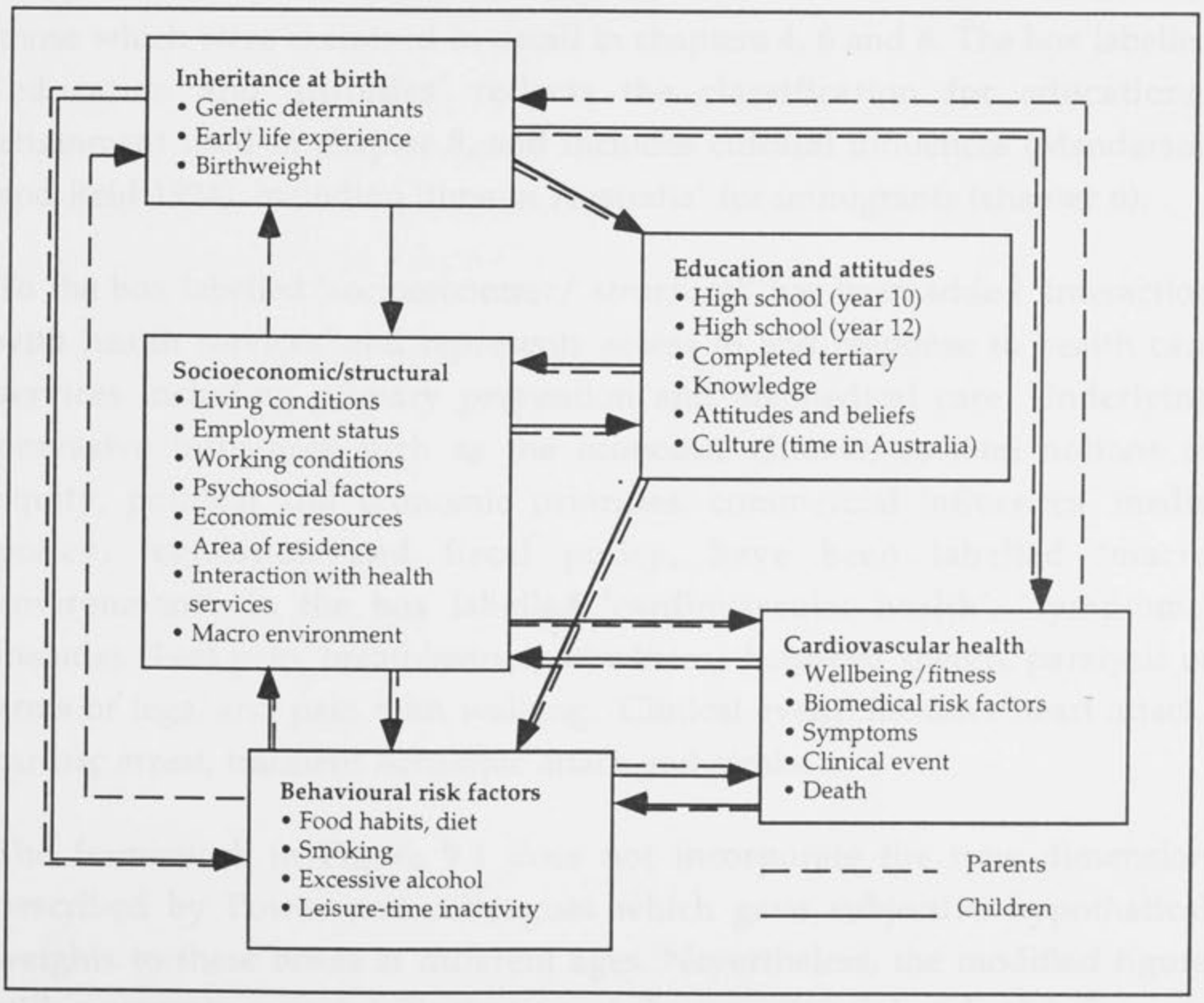

FIGURE 9.1 ***Inter- and intragenerational relationships between cardiovascular health and circumstances.***

The authors described their diagram as a 'gross oversimplification' noting that it did not adequately represent the relationships between the factors that were boxed together; that the factors in each box were not exhaustive; that the suggested relationships between boxes did not imply relationships between all factors within those boxes; that most factors change over time as the individual ages and passes through the life cycle; and that only a limited range of health outcomes were included. While these limitations apply equally to the modified version, the diagram helps to crystallise some of the influences and relationships which may have led to the trends and persistent social inequalities demonstrated in chapters 4 to 8.

The box labelled 'inheritance at birth' has been modified to better reflect the increasing evidence for the role of genetic determinants, and the work of Barker and colleagues concerning the role of deprivation in early life. Lines have been added to represent the relationship between 'inheritance at birth' and 'behavioural risk factors'. The characteristics listed under 'socioeconomic/structural' are mostly those discussed in chapter 2 under 'materialist and structural explanations', and are especially relevant to the analysis of occupation groups in chapter 7. The 'behavioural risk factors' are those which were examined in detail in chapters 4, 6 and 8. The box labelled 'education and attitudes' reflects the classification for educational attainment used in chapter 8, and includes cultural influences (Manderson and Reid 1994), including 'time in Australia' for immigrants (chapter 6).

To the box labelled 'socioeconomic/ structural' has been added 'interaction with health services' and represents access to and response to health care services including primary prevention and all medical care. Underlying pervasive influences such as the economic climate, societal notions of equity, political and economic priorities, commercial influences, media policy, legislation and fiscal policy, have been labelled 'macro environment'. In the box labelled 'cardiovascular health', `symptoms' includes chest pain, breathlessness, tiredness, disturbed speech, paralysis of arms or legs, and pain with walking. 'Clinical event' includes heart attack, cardiac arrest, transient ischaemic attack and stroke.

The framework in Figure 9.1 does not incorporate the time dimension described by Power and colleagues which gave subjective hypothetical weights to these boxes at different ages. Nevertheless, the modified figure still represents a useful illustration of the complex network of influences behind cardiovascular disease and its outcomes.

9.2 Trends and social inequalities

Trends in risk factors

Chapter 4 showed that the risk factor profile of Australian adults generally improved during the 1980s. Blood pressure levels declined in all age-sex groups and hypertension appeared to be more effectively managed. Adding salt to food became less common as did eating the fat on meat. The prevalence of smoking and consumption of alcohol declined significantly in both men and women. Total cholesterol levels decreased significantly in younger men and older women. Walking for recreation or exercise and other forms of less vigorous exercise became more popular.

There were some important exceptions to the overall improvement. Lipid results generally showed no overall favourable trend; the prevalence of aerobic exercise and vigorous exercise did not change; and, the most disturbing finding, weight for height increased in all ages strongly suggesting increased body fatness.

It is concluded that reductions in cigarette smoking and blood pressure are likely to have contributed to the falls in cardiovascular mortality.

Changes in self-reported dietary behaviour were consistent with health education messages to avoid salt and fat on meat, although the trend towards greater body fatness indicates that net change in overall dietary behaviour has not been effective in maintaining a healthy weight.

The trend towards greater body fatness requires greater attention because it may retard the benefits of favourable trends in other cardiovascular risk factors, and eventually impact on morbidity and mortality.

Effect of blood pressure measurement error on trend estimates

Chapter 5 showed that last digit preference for zero may inflate estimates of proportions having high blood pressure. A tendency to record identical duplicate measurements could contribute 0.85 mmHg to time trends or geographic differences in mean systolic blood pressure (but not diastolic blood pressure). The latter phenomenon contributed approximately 12% to the estimated falls in systolic blood pressure observed in Australian adults during the 1980s.

It is concluded that the observed falls represent real decreases in the average level of systolic blood pressure among Australian adults.

It is recommended that epidemiological studies for geographic and trend differentials in systolic blood pressure be mindful of the effects of measurement error in their analysis. Training procedures for blood pressure measurement are of critical importance if measurement error is to be minimised. Adherence to the measurement protocol should be monitored continuously during data collection to ensure comparability of results in multi-centre studies.

Risk factor differentials in immigrants

Chapter 6 identified significant differences between immigrant groups and Australian-born men and women, particularly for systolic blood pressure, overall obesity and behavioural risk factors. There were few substantial differences in blood lipid concentrations, and little evidence to suggest that total plasma cholesterol has played a major role in lower cardiovascular mortality among immigrants. Important differences were demonstrated between the risk factor profiles of immigrant groups which originated from the same geographic region, particularly immigrant groups from Britain, from Southern Europe and from Asia. The acculturation process affected immigrant groups differently. Generally, systolic blood pressure increased with period of residency in Australia. Body mass index increased among Asian immigrants, as did participation in physical activity during leisure-time.

It is concluded that risk factors commonly accepted as determinants of cardiovascular disease are an insufficient explanation of the lower standardised mortality ratios from cardiovascular disease among most immigrant groups in Australia. Systolic blood pressure best explained variation in cardiovascular mortality among male immigrants, and smoking prevalence proved important among female immigrants.

Factors other than the traditional risk factors are required to explain the relatively low cardiovascular mortality rates among those immigrant groups with a relatively poor risk factor profile (eg. immigrants from Greece and men from the Middle East). The immigration selection process is one possibility. Others factors suggested by Figure 9.1 which may have a positive influence on cardiovascular health include 'inheritance at birth', factors not identified in this study possibly mediated through dietary pathways, and beneficial psychosocial factors such as strong social support networks.

Trends in socioeconomic inequalities in mortality

Chapter 7 showed that socioeconomic inequalities in coronary mortality among men[1] continued to widen during the early 1980s, but stabilised thereafter and persisted into the nineties. During the 1980s, men in manual occupations were at least 35% more likely to die from coronary heart disease than men in professional occupations, and 60% more likely to die from stroke. Their 5-year population risk of a coronary event was 30% higher due to higher systolic blood pressure and greater smoking rates. Both groups experienced reductions in coronary risk and mortality during that decade.

It is concluded that differentials in blood pressure and smoking prevalence have contributed to socioeconomic differentials in coronary mortality, and that trends in these same risk factors have contributed to declines in coronary mortality in each socioeconomic strata. This is also likely to be true for stroke.

The results also suggest that the often quoted observation 'when mortality is declining, socioeconomic differentials for heart diseases tend to increase' (Yeracaris and Kim 1978), may need to be supplemented, at least in Australia, with the words *'initially but stabilise thereafter.'*

Trends in socioeconomic inequalities in risk factors

Chapter 8 identified beneficial and detrimental trends in risk factors for different socioeconomic groups. During the 1980s, average blood pressure declined for each level of educational attainment. Dietary messages to reduce the intake of saturated fat had little apparent effect on the lipid profile of any population group. There was a strong association between adult height and educational attainment, possibly reflecting socioeconomic differentials in nutrition and health during childhood. The average weight of women increased significantly (by up to 4.4 kg) depending on age and educational attainment. Increases were most pronounced in women of lower educational attainment. The average weight of older men increased by about 2.5 kg regardless of educational attainment. Advice to avoid salt was adopted across the spectrum of educational attainment but with no suggestion that the socioeconomic gradient, which favoured the more highly educated, was diminishing.

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[1] Data limitations precluded the analysis for women.

Men of all education levels responded positively to anti-smoking initiatives but the relative disadvantage of those of lower education was maintained. Among women, the decline in smoking was less among those in the low education group which suggests a widening socioeconomic gap in smoking prevalence. The prevalence of moderate to heavy drinkers was higher among men of lower educational attainment but declined significantly over the period. Walking for recreation or exercise became more popular, especially among older men of low education, while the prevalence of aerobic exercise and vigorous exercise remained largely unchanged. Overall, the clear socioeconomic gradient between leisure-time physical activity and educational attainment remained.

It is concluded that the lower socioeconomic group has improved its risk factor profile but its relative disadvantage compared with the higher socioeconomic group persists. Health promotion activities in Australia seem to have reached and influenced the lower socioeconomic groups but the challenge to reduce inequalities remains. The steady increase in educational attainment in Australia may have been an important factor in the general improvement in the nation's risk factor profile and in the decrease in mortality from coronary heart disease.

9.3 Discussion and recommendations

While observational studies such as the Risk Factor Prevalence Study have an important monitoring role and can contribute to an assessment of prevention activities, their ability to explore the causal links in Figure 9.1 is limited. However, several findings deserve to be examined more fully even though the discussion has to be somewhat tentative. First, the opposing trends in blood pressure and body fatness warrant special comment.

The marked falls in blood pressure

The marked decline in blood pressure that occurred at all levels of society is one of the more important observations. The causes of the decline over time are not well understood. Improved management of hypertension is an insufficient explanation because Australians not on anti-hypertensive medication (most of the population) also experienced significant declines in average blood pressure. The sizes of the decreases were too great to be explained by measurement error. The observed increases in body mass index eliminate weight reduction as a plausible explanation. Participation in exercise during leisure-time showed little change except for an increase in the prevalence of self-reported walking, and it is plausible that this may

have contributed in part to the fall in blood pressure. Changes in the national diet may be an explanation if it is assumed that the questions on dietary behaviour concerning salt and fat are broad indicators of healthy dietary behaviour. However, there was little change in the lipid profile and body mass index increased. Thus, although the socioeconomic gradients in body mass index, exercise and dietary behaviour are consistent with the socioeconomic gradient in blood pressure these risk factors offer few clues as to the explanation of the decline in blood pressure over time.

The trend towards increased body fatness

The prevalence of overweight and obesity is increasing among Australian adults, as it is in other developed countries, and concern has been expressed about the public health implications of this trend. This thesis has demonstrated a strong inverse gradient between body fatness and educational attainment, and significant increases over time in body fatness at every level of educational attainment. One explanation for this pattern is that exposure to formal education in the past had a direct effect and, as a consequence, the more highly educated people were less likely to overeat and be inactive, but that now all strata are exposed to external influences that lead to increased body fatness. Certainly, the evidence suggests that Australians are presently eating too much and exercising too little at all levels of society.

It may also be that educational attainment is an indicator of social and environmental factors experienced in early life and the differentials in part reflect the degree to which behaviour and eating patterns of adults are set in childhood. Unfortunately, the cross-sectional nature of the Risk Factor Prevalence Study does not allow the dynamics of the relationship between education and body fatness to be fully explored. This would require the analysis of longitudinal data which takes account of socioeconomic status of origin as well as adulthood.

To determine ways of tackling the socioeconomic inequalities, it is necessary to understand the determinants of obesity as well as the consequences. The relationship between obesity and socioeconomic status is powerful, complex and probably bidirectional (Stunkard and Sorensen 1993). Whilst it has been known that socioeconomic status influences obesity, recent evidence also suggests that obesity influences socioeconomic status (Gortmaker et al. 1993). That is, obese adolescents (girls in particular), may be destined for poorer socioeconomic status in later life through selective social mobility, irrespective of their social background (Carpenter and Bartley 1994). This

process may involve discrimination against overweight persons (Gortmaker et al. 1993). Thus, the inverse socioeconomic gradient in overweight is likely to partly reflect the socioeconomic consequence of being overweight as well as the influence of socioeconomic status, principally through behavioural factors such as diet and exercise (Gortmaker et al. 1993). However, it may also be that psychological and emotional factors influence both weight and socioeconomic status in adult life and that the association between overweight and socioeconomic status is the result of these common influences. To complicate the matter further, there is also evidence that both socioeconomic status and overweight are influenced by heredity factors (Teasdale et al. 1990). It seems therefore that structural, social and educational strategies will be needed if the trend towards greater body fatness is to be reversed and the socioeconomic inequalities reduced. Jeffery (1991) believes it is time to think of alternatives to the traditional individual-focused strategies. The food supply, it is suggested, should be viewed as a potential environmental hazard that promotes obesity and which requires public health policy strategies to improve it. On the other side of the equation, there are different subgroups of the inactive who may require different public health strategies to encourage them to adopt regular exercise habits (Owen and Bauman 1992; Siegel et al. 1995). The greatest health benefits to the community are likely to be achieved by activating the sedentary rather than by increasing the activity levels of those already exercising.

Behaviour change and socioeconomic status

This thesis has demonstrated that the major behaviours known to be associated with increased risk of cardiovascular disease are also strongly associated with educational attainment. Cigarette smoking, moderate to heavy drinking, leisure-time inactivity, and 'unhealthy' dietary practices were each shown to be more prevalent among those of lower educational attainment. These behaviours were also shown to occur together more often in those of lower education. This much is already known. What has also been demonstrated is that significant behaviour change can occur simultaneously across the socioeconomic spectrum (in this case defined by educational attainment) of a free-living society, and not primarily in higher socioeconomic groups. The degree to which these behaviours are 'voluntary' may vary between strata. Some of the barriers to behaviour change were discussed in chapter 2. It is easier for individuals to adopt healthier lifestyles when such behaviours become socially desirable or, in the case of smoking, less acceptable (Oberman et al. 1994). This may well

have been the case in Australia. On the other hand, there is little evidence of diminishing differences in risk factor behaviours between educational groups, even though reducing such inequalities is a major goal of public health policy in Australia. This general pattern is consistent with the proposition that it is the higher socioeconomic groups in society who are first to 'respond' to health education messages, and this generates socioeconomic inequalities, but a 'trickle-down effect' eventually brings benefit also to the lower socioeconomic strata.

Implications for health promotion

More broadly, it appears that cardiovascular risk and mortality have declined over the past decade in Australia and that differentials between socioeconomic groups have been relatively stable. What are the implications of this for the presumed success of health promotion and its future directions? It has been argued that health education and improvements in health related behaviour benefit higher social classes more than lower classes, possibly acting to increase health differentials (Blaxter 1991). The results of this study are not inconsistent with this proposition, but the Australian findings also suggest that, in relation to cardiovascular disease:

- community based health promotion is of benefit to all socioeconomic strata, in time;
- while there might be a lag in uptake between socioeconomic strata that generates inequalities, similar trends are experienced once uptake begins; and
- socioeconomic differentials are persistent and require alternative strategies to those implemented so far if they are to be diminished.

It may be that *absolute* improvements in overall cardiovascular health are greater and achieved faster by broadly based community approaches even though they tend to generate and perpetuate the *relative* inequalities between socioeconomic groups.

The results also suggest that strategies to improve the blood lipid profile of Australians have the potential to yield additional cardiovascular benefits for all age, sex socioeconomic strata (since there was little improvement during the 1980s).

Recommendations for future monitoring

The monitoring of trends and inequalities in cardiovascular risk factors and mortality is an important component of an integrated monitoring system

for cardiovascular disease. The risk factor prevalence surveys conducted during the 1980s by the National Heart Foundation of Australia provided valuable data on physical measurements such as blood pressure and blood lipids which were unavailable from any other source. However, the surveys did not include rural areas or cover risk factors in the young or elderly. The survey program does not have committed funding and a further survey is not scheduled at this stage. Future surveys using the same protocol would provide very useful trend data, especially for biomedical risk factors, but the uncertainty of the inter-survey period limits its usefulness for regular monitoring purposes. Alternative strategies for collecting regular biomedical risk factor data need to be developed.

Anthropometric measurements and blood pressure are being collected as part of the nutrition component of the National Health and Nutrition Survey which is being conducted by the Australian Bureau of Statistics during 1995 and this will address some of the data gaps which exist at present. As a monitoring mechanism for cardiovascular disease however, the survey has its limitations. Blood sampling is not included, the nutrition component is unlikely to be repeated for at least ten years, and the content of the health component, which is conducted every five years and collects self-reported risk factor behaviour data, is subject to competing demands for questionnaire space and interviewer time. Perhaps the conduct of large and complex surveys at five-yearly intervals is not the most effective way of monitoring the health and nutritional status of the population. The option of a program of continuous data collection, with the possibility of a longitudinal component, has been adopted in some countries and it may be time to examine the efficacy of such an approach in the Australian context.

Regarding mortality data, the death registration system is an important data source for epidemiological analysis and it is important that the quality of the data is high. The validity and coverage of the demographic descriptors need to be examined, especially occupation but also area of residence, birthplace and length of stay. For cardiovascular disease, there is the need to validate causes of death other than coronary heart disease.

More systematic use could be made of the routinely collected mortality data for monitoring trends in inequalities in cardiovascular mortality. The analysis in this thesis (chapter 7) is the first to examine trends in socioeconomic inequalities in mortality data after 1969-78 possibly because of the difficulties in analysis posed by the non-simultaneous introduction of a new occupation coding system into the mortality, labour force and census

collections. Clearly the vital and demographic data collection systems are intimately related and future changes need to be coordinated to maximise compatibility. More broadly, this thesis has clearly demonstrated that monitoring inequalities and the epidemiological search for their causes require data which span social, economic, demographic, vital and health collection systems. Such analyses are greatly facilitated by uniformity and consistency across data collection systems in data definition and coding systems. This need is likely to receive added impetus if record linkage for epidemiological purposes becomes more common in Australia.

Record linkage has the potential to enhance the usefulness of routinely collected data and to provide outcome data for longitudinal studies in a cost effective and efficient manner. Record linkage could also assist in examining socioeconomic factors associated with inequalities in cardiovascular events and risk factors since it is not practical to collect a comprehensive set of sociodemographic variables in all data collections. One limitation of the National Heart Foundation's Risk Factor Prevalence Study is the lack of outcome data. The value of the survey datasets would have been enhanced had it been possible to link the risk factor data to subsequent hospital morbidity and death.

Social justice and future monitoring of cardiovascular disease

A recent Federal government report (Commonwealth Department of Human Services and Health 1994) has identified, as part of the national health goals and target process, the need to establish and maintain a national monitoring system for cardiovascular disease, its risk factors and management. Components of an integrated monitoring system are seen to include predisposing factors, incidence, pre-hospital and emergency care, medical and surgical care, rehabilitation, follow-up care, and palliative care in addition to risk factors and mortality. The principle of social justice has been incorporated into the national health goals and target process through the goal of reducing the level of health inequalities in Australia and two of the three priority populations identified are socioeconomically disadvantaged Australians and people of non-English speaking backgrounds. Both groups have been the focus of this thesis and the results provide the rationale for the continued monitoring of trends and inequalities and the development of strategies to achieve social justice in cardiovascular health.

References

Abbott RD, Rodriguez BL, Burchfiel CM and Curb JD (1994) Physical activity in older middle-aged men and reduced risk of stroke: the Honolulu Heart Program. *Am J Epidemiol* 139:881-893.

Ajzen I and Fishbein M (1980) Understanding attitudes and predicting social behaviour. Prentice-Hall, Englewood Cliffs, NJ.

Akerstedt T, Knutsson A, Alfredsson L and Theorell T (1984) Shift work and cardiovascular disease. *Scand J Work Environ Health* 10:409-414.

Al-Roomi KA, Dobson AJ, Hall E, Heller RF and Magnus P (1989) Declining mortality from ischemic heart disease and cerebrovascular disease in Australia. *Am J Epidemiol* 129:503-510.

Al-Roomi KA, Heller RF and Wlodarczyk J (1990) Hypertension control and the risk of myocardial infarction and stroke: a population-based study. *Med J Aust* 153:595-603.

Al-Roomi K, Heller RF, Holland T, Floate D and Wlodarczyk J (1992) The importance of hypertension in the aetiology of infarctive and haemorrhagic stroke. The Lower Hunter Stroke Study. *Med J Aust* 157:452-455.

Alfredsson L, Karasek R and Theorell T (1982) Myocardial infarction risk and psychosocial work environment: an analysis of the male Swedish working force. *Soc Sci Med* 16:463-467.

Anonymous (1982) Prevention of rheumatic heart disease [editorial]. *Lancet* i:142-143.

Anonymous (1994) Essence of stress [editorial]. *Lancet* 344:1713-1714.

Antonovsky A (1989) Social inequalities in health: a complementary perspective. *In:* Fox J (ed) *Health inequalities in European countries.* Gower, Aldershot.

Armitage P, Fox W, Rose GA and Tinker CM (1966) The variability of measurements of casual blood pressure: II. Survey experience. *Clin Sci* 30:337-344.

Armstrong BK, Margetts BM, Masarei JRL and Hopkins SM (1983) Coronary risk factors in Italian migrants to Australia. *Am J Epidemiol* 118:651-658.

Arroll B and Beaglehole R (1992) Does physical activity lower blood pressure: a critical review of the clinical trials. *J Clin Epidemiol* 45:439-447.

Assmann G and Schulte H (1992) Relation of high-density lipoprotein cholesterol and triglycerides to incidence of atherosclerotic coronary artery disease (the PROCAM experience). Prospective Cardiovascular Munster study. *Am J Cardiol* 70:733-737.

Atkins D, Psaty BM, Koepsell TD, Longstreth WT Jr and Larson EB (1993) Cholesterol reduction and the risk for stroke in men. A meta-analysis of randomized, controlled trials. *Ann Intern Med* 119:136-145.

Australian Bureau of Statistics (1987) *1986 Census of population and housing - microfiche CX0003*. AGPS, Canberra.

Australian Bureau of Statistics (1988a) *Australian Standard Classification of Occupations/ Classification and Classified List of Occupations: Link. Cat. No. 2182.0*. ABS, Canberra.

Australian Bureau of Statistics (1988b) *Australian Standard Classification of Occupations/ Classification and Classified List of Occupations: Link file. Technical details. Cat. No. 2199.0*. ABS, Canberra.

Australian Bureau of Statistics (1990) *Australian standard classification of countries for social statistics. Cat. No. 1269.0*. ABS, Canberra.

Australian Bureau of Statistics (1991) *1989-90 National Health Survey. Cardiovascular and related conditions. Cat. No. 4272.0*. ABS, Canberra.

Australian Bureau of Statistics (1992a) *1989-90 National Health Survey: Smoking, Australia. Cat. No. 4382.0*. ABS, Canberra.

Australian Bureau of Statistics (1992b) *1989-90 National Health Survey. Health status indicators, Australia. Cat. No. 4370.0*. ABS, Canberra.

Australian Bureau of Statistics (1994a) *1989-90 National Health Survey, Lifestyle and health, Australia. Cat. No. 4366.0*. ABS, Canberra.

Australian Bureau of Statistics (1994b) Education. *In: Australian Social Trends 1994. Cat. No. 4102.0*. ABS, Canberra.

Australian Institute of Health and Welfare (1992) *Australia's health 1992: the third biennial report of the Australian Institute of Health and Welfare*. AGPS, Canberra. p. 212.

Australian Institute of Health and Welfare (1994) *Australia's health 1994: the fourth biennial report of the Australian Institute of Health and Welfare*. AGPS, Canberra.

Baker D, Illsley R and Vågerö D (1993) Today or in the past? The origins of ischaemic heart disease. *J Public Health Med* 15:243-248.

Baker D (1994) Poverty and ischaemic heart disease: the missing links [comment]. *Lancet* 343:496.

Barker DJP (1992) The maternal and fetal origins of cardiovascular disease. *J Epidemiol Community Health* 46:8-11.

Barker DJP and Osmond C (1992) The maternal and infant origins of cardiovascular disease. *In:* Marmot M and Elliott P (eds) *Coronary heart disease epidemiology. From aetiology to public health*. Oxford University Press, Oxford. p. 83-90.

Barker DJP, Gluckman PD, Godfrey KM, Harding JE, Owens JA and Robinson JS (1993) Fetal nutrition and cardiovascular disease in adult life. *Lancet* 341:938-941.

Barker DJP (1995) Fetal origins of coronary heart disease. Br Med J 311:171-174.

Bauman A, Owen N and Rushworth RL (1990) Recent trends and socio-demographic determinants of exercise participation in Australia. *Community Health Stud* 14:19-26.

Baxter J, Emmison M, Western J and Western M (1991) Class analysis and contemporary Australia. MacMillan, Melbourne.

Beaglehole R, Dobson A, Hobbs MST, Jackson R and Martin CA (1989) CHD in Australia and New Zealand. *Int J Epidemiol* 18(Suppl 1):S145-S148.

Beaglehole R (1990) International trends in coronary heart disease mortality, morbidity, and risk factors. *Epidemiol Rev* 12:1-15.

Beilin LJ and Puddey IB (1992) Alcohol and hypertension. *Clin Exp Hypertens* A 14:119-138.

Bengtsson C, Björkelund C, Lapidus L and Lissner L (1993) Associations of serum lipid concentrations and obesity with mortality in women: 20 year follow-up of participants in prospective population study in Gothenburg, Sweden. *Br Med J* 307:1385-1388.

Bennett S, Donovan J, Stevenson C and Wright P (1994) *Mortality surveillance, Australia 1981-1992. Australian Institute of Health and Welfare: Mortality Surveillance Series, No. 2.* AGPS, Canberra.

Bennett SA (1995) Cardiovascular risk factors in Australia: trends in socioeconomic inequalities. *J Epidemiol Community Health* 49:294-313.

Benotti PN, Bistrian B, Benotti JR, Blackburn G and Forse RA (1992) Heart disease and hypertension in severe obesity: the benefits of weight reduction. *Am J Clin Nutr* 55(Suppl 2):S586-S590.

Ben-Shlomo Y and Davey Smith G (1991) Deprivation in infancy or in adult life: which is more important for mortality risk? *Lancet* 337:530-534.

Bergstrand R, Vedin A, Wilhelmsson C and Wilhelmsen L (1983) Bias due to non-participation and heterogenous sub-groups in population surveys. *J Chron Dis* 36:725-728.

Berkman LF and Syme L (1979) Social networks, host resistance, and mortality: a nine-year follow-up study of Alameda county residents. *Am J Epidemiol* 109:186-204.

Berlin JA and Colditz GA (1990) A meta-analysis of physical activity in the prevention of coronary heart disease. *Am J Epidemiol* 132:612-628.

Björntorp P (1988) The associations between obesity, adipose tissue distribution and disease. *Acta Med Scand* 723(Suppl):121-134.

Björntorp P (1992) Abdominal fat distribution and disease: an overview of epidemiological data. *Ann Med* 24:15-18.

Black D (1980) *Inequalities in health: Report of a Working Group.* DHSS, HMSO, London.

Blackburn H and Luepker R (1986) Heart disease. *In: Maxcy-Rosenau Public Health and Preventive Medicine.* Appleton-Century-Crofts, Norwalk, Connecticut. p. 1159-1193.

Blackburn H, Watkins LO, Agras WS, Cartleton RA and Falkner B (1987) Primary prevention of coronary heart disease. *Circulation* 76(Suppl I):164-167.

Blackburn H (1989) Trends and determinants of CHD mortality: Changes in risk factors and their effects. *Int J Epidemiol* 18:S210-S215.

Blair SN, Goodyear NN, Gibbons LW and Cooper KH (1984) Physical fitness and incidence of hypertension in healthy normotensive men and women. *JAMA* 252:487-490.

Blair SN, Kohl HW III, Paffenbarger RS Jr, Clark DG, Cooper KH and Gibbons LW (1989) Physical fitness and all-cause mortality. A prospective study of healthy men and women. *JAMA* 262:2395-2401.

Blair SN, Kohl HW, Gordon NF and Paffenbarger RS Jr (1992) How much physical activity is good for health? *Annu Rev Publ Health* 13:99-126.

Blair SN (1993) Evidence for success of exercise in weight loss and control. *Ann Intern Med* 119:702-706.

Blair SN, Shaten J, Brownell K, Collins G and Lissner L (1993) Body weight change, all-cause mortality, and cause-specific mortality in the Multiple Risk Factor Intervention Trial. *Ann Intern Med* 119:749-757.

Blane D, Smith GD and Bartley M (1990) Social class differences in years of potential life lost: size, trends, and principal causes. *Br Med J* 301:429-432.

Blane D, Smith GD and Bartley M (1993) Social selection: what does it contribute to social class differences in health? *Sociol Health Illn* 15:1-15.

Blaxter M (1987) Evidence on inequality in health from a national survey. *Lancet* 2:30-33.

Blaxter M (1990) *Health and lifestyles*. Tavistock/Routledge, London.

Blaxter M (1991) Fifty years on - Inequalities in health. *In:* Murphy M and Hobcraft J (eds) *Population research in Britain. Supplement to Population Studies Vol. 45.* University Press, Cambridge. p. 69-94.

Boyle CA, Dobson AJ, Egger G and Magnus P (1994) Can the increasing weight of Australians be explained by the decreasing prevalence of cigarette smoking? *Int J Obesity* 18:55-60.

Boyle P and Parkin DM (1991) Statistical methods for registries. *In:* Jensen OM, Parkin DM, MacLennan R, Muir CS and Skeet RG (eds) *Cancer registration principles and methods, Scientific Publication No. 95.* International Agency for Research on Cancer, Lyon.

Brannstrom I, Weinehall L, Persson LA, Wester PO and Wall S (1993) Changing social patterns of risk factors for cardiovascular disease in a Swedish community intervention programme. *Int J Epidemiol* 22:1026-1037.

Breslow NE and Day NE (1987) *Statistical methods in cancer research. II. The design and analysis of cohort studies.* IARC Scientific Publications No. 82, Lyon. p. 120-176.

Britton M (ed) (1990) *Mortality and geography. A review in the mid-1980s, England and Wales.* OPCS, HMSO, London.

Brownell KD and Wadden TA (1992) Etiology and treatment of obesity: understanding a serious, prevalent, and refractory disorder. *J Consult Clin Psychol* 60:505-516.

Bruce NG, Shaper AG, Walker M and Wannamethee G (1988) Observer bias in blood pressure studies. *J Hypertension* 6:375-380.

Brundtland GH (1994) Influencing environmental factors in cardiovascular disease prevention - a global view. *Prev Med* 23:531-534.

Bureau of Immigration Research (1992) *Immigration update, December quarter 1991.* AGPS, Canberra.

Calnan M (1989) Control over health and patterns of health related behaviour. *Soc Sci Med* 29:131-136.

Calnan M and Williams S (1991) Style of life and the salience of health: an exploratory study of health related practices in households from differing socioeconomic circumstances. *Soc Hlth Illns* 13:506-529.

Canner PL, Borhani NO, Oberman A, et al. (1991) The hypertension prevention trial: assessment of the quality of blood pressure measurements. *Am J Epidemiol* 134:379-392.

Carleton RA, Dwyer J, Finberg L, et al. (1991) Report of the Expert Panel on Population Strategies for Blood Cholesterol Reduction. A statement from the National Cholesterol Education Program, National Heart, Lung and Blood Institute. *Circulation* 83:2182.

Carpenter L and Bartley M (1994) Fat, female, and poor. *Lancet* 344:1715-1716.

Carr-Hill RA (1988) Time trends in inequalities in health. *J Biosoc Sci* 20:265-273.

Carr-Hill R (1990) The measurement of inequities in health: lessons from the British experience. *Soc Sci Med* 31:393-404.

Carter WB (1991) Health behaviour as rational process: theory of reasoned action and multiattribute utility theory. *In:* Glanz K, Lewis FM and Rimer BK (eds) *Health behaviour and health education.* Jossey-Bass, Oxford.

Cashel K, English R, Bennett S, Berzins J, Brown G and Magnus P (1986) *National dietary survey of adults: 1983. No. 1 Foods consumed*. Commonwealth Department of Health, AGPS, Canberra.

Castelli W and Leaf A (1985) Identification and assessment of cardiac risk - an overview. *Cardiol Clin* 3:171-178.

Chambless LE, Dobson AJ, Patterson CC and Raines B (1990) On the use of a logistic risk score in predicting risk of coronary heart disease. *Stat Med* 9:385-396.

Chen Z, Peto R, Collins R, MacMahon S, Lu J and Li W (1991) Serum cholesterol concentration and coronary heart disease in a population with low cholesterol concentrations. *Br Med J* 303:276-282.

Choi SC, Boxerman SB and Steinburg L (1978) Nurses' preference of terminal digits in data reading. *J Nurs Educ* 17:38-41.

Chun BY, Dobson AJ and Heller RF (1993) Smoking and the incidence of coronary heart disease in an Australian population. *Med J Aust* 159:508-512.

Clausen J and Jensen G (1992) Blood pressure and mortality: an epidemiological survey with 10 years follow-up. *J Hum Hypertens* 6:53-59.

Collins R, Peto R, MacMahon S, et al. (1990) Blood pressure, stroke, and coronary heart disease. Part 2, short-term reductions in blood pressure: overview of randomised drug trials in their epidemiological context. *Lancet* 335:827-838.

Commonwealth Department of Health (1982) *Dietary guidelines for Australians.* AGPS, Canberra.

Commonwealth Department of Human Services and Health (1994) *Better health outcomes for Australians. National goals, targets and strategies for better health outcomes into the next century.* AGPS, Canberra.

Consensus Panel (1994) The management of hypertension: a consensus statement. *Med J Aust* 160(Suppl):S1-S16.

Criqui MH, Barrett-Connor E and Austin M (1978) Differences between respondents and non-respondents in a population-based cardiovascular disease study. *Am J Epidemiol* 108:367-372.

Criqui MH and Ringel B (1994) Does diet or alcohol explain the French paradox? *Lancet* 344:1719-1723.

Cullen KJ, Knuiman MW and Ward NJ (1993) Alcohol and mortality in Busselton, Western Australia. *Am J Epidemiol* 137:242-248.

Czarn AOS, Jamrozik K, Hobbs MST and Thompson PL (1992) Follow-up care after acute myocardial infarction. *Med J Aust* 157:302-305.

Dasvarma GL (1980) *Differential mortality in Australia 1970–72 (PhD Thesis).* Department of Demography, Australian National University, Canberra.

Davey Smith G and Shipley MJ (1990) Magnitude and causes of socioeconomic differentials in mortality: further evidence from the Whitehall study. *J Epidemiol Community Health* 44:265-270.

De Busk RF, Stenestrand U, Sheehan M and Haskell WL (1990) Training effects of long versus short bouts of exercise in healthy subjects. *Am J Cardiol* 64:1010-1013.

De Gaudemaris R, Folsom AR, Prineas RJ and Luepker RV (1985) The random-zero versus the standard mercury sphygmomanometer: a systematic blood pressure difference. *Am J Epidemiol* 121:282-290.

d'Espaignet ET (1994) *Trends in Australian mortality: Diseases of the circulatory system, 1950–1991. Australian Institute of Health and Welfare: Mortality Series, No. 2.* AGPS, Canberra.

Dischinger P and DuChene AG (1986) Quality control aspects of blood pressure measurements in the Multiple Risk Factor Intervention Trial. *Controlled Clin Trials* 7(Suppl 3):137S-157S.

Dobson AJ, Gibberd RW, Wheeler DJ and Leeder SR (1981) Age-specific trends in mortality from ischemic heart disease and cerebrovascular disease in Australia. *Am J Epidemiol* 113:404-412.

Dobson AJ, Gibberd RW and Leeder SR (1983) Death certification and coding for ischemic heart disease in Australia. *Am J Epidemiol* 117:397-405.

Dobson AJ, Gibberd RW, Leeder SR and O'Connell DL (1985) Occupational differences in ischemic heart disease mortality and risk factors in Australia. *Am J Epidemiol* 122:283-290.

Dobson AJ (1987) Trends in cardiovascular risk factors in Australia, 1966-1983: evidence from prevalence surveys. *Community Health Stud* 11:2-14.

Dobson AJ, Gibberd RW, Leeder SR, Alexander HM, Young AF and Lloyd DM (1988) Ischemic heart disease in the Hunter region of New South Wales, Australia, 1979-1985. *Am J Epidemiol* 128:106-115.

Dobson AJ, Alexander HM, Heller RF and Lloyd DM (1991a) How soon after quitting smoking does risk of heart attack decline? *J Clin Epidemiol* 44:1247-1253.

Dobson AJ, Alexander HM, Heller RF, Malcolm JA and Steele PL (1991b) Trends in ischaemic heart disease in the Hunter Region of New South Wales 1985-1989. *Med J Aust* 155:599-608.

Dobson A, Halpin S and Alexander H (1991c) Does the occupational structure of the Hunter Region explain the high rates of ischaemic heart disease among its men? *Aust J Public Health* 15:172-177.

Donahue RP, Abbott RD, Reed DM and Yano K (1986) Alcohol and hemorrhagic stroke. The Honolulu Heart Program. *JAMA* 255:2311-2314.

Dunt DR (1982) Recent mortality trends in the adult Australian population and its principal ethnic groupings. *Community Health Stud* 6:217-222.

Dwyer T and Hetzel BS (1980) A comparison of trends of coronary heart disease mortality in Australia, USA and England and Wales with reference to three major risk factors - hypertension, cigarette smoking and diet. *Int J Epidemiol* 9:65-71.

Eilersten E and Humerfelt S (1968) The observer variation in the measurement of arterial blood pressure. *Acta Med Scand* 184:145-157.

Ekelund L-G, Haskell WL, Johnson JL, Whaley FS, Criqui MH and Sheps DS (1988) Physical fitness as a predictor of cardiovascular mortality in asymptomatic North American men. The Lipid Research Clinics Mortality Follow-up Study. *N Engl J Med* 319:1379-1384.

Elford J, Whincup P and Shaper AG (1991) Early life experience and adult cardiovascular disease: longitudinal and case-control studies. *Int J Epidemiol* 20:833-844.

Elford J, Shaper AG and Whincup P (1992) Early life experience and cardiovascular disease - ecological studies. *J Epidemiol Community Health* 46:1-8.

English R and Bennett S (1985) Overweight and obesity in the Australian community. *J Food Nutr* 42:2-12.

English R, Cashel K, Bennett S, Berzins J, Waters A-M and Magnus P (1987) *National dietary survey of adults: 1983. No. 2 Nutrient intakes.* Commonwealth Department of Community Services and Health, AGPS, Canberra.

Epstein FH (1989) The relationship of lifestyle to international trends in CHD. *Int J Epidemiol* 18(Suppl 1):S203-S209.

Ernsberger P and Koletsky RJ (1993) Weight cycling and mortality: support from animal studies. *JAMA* 269:1116.

Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (1993) Summary of the Second Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. (Adult Treatment Panel II). *JAMA* 269:3015-3023.

Fall CHD, Vijayakumar M, Barker DJP, Osmond C and Duggleby S (1995a) Weight in infancy and prevalence of coronary heart disease in adult life. *Br Med J* 310:17-19.

Fall CHD, Osmond C, Barker DJP, Clark PMS, Hales CN, Stirling Y and Meade TW (1995b) Fetal and infant growth and cardiovascular risk factors in women. *Br Med J* 310:428-432.

Feinstein JS (1993) The relationship between socioeconomic status and health: a review of the literature. *Milbank Q* 71:279-322.

Feldman JJ, Makuc DM, Kleinman JC and Cornoni-Huntley J (1989) National trends in educational differentials in mortality. *Am J Epidemiol* 129:919-933.

Fletcher GF, Blair SN, Blumenthal J, et al. (1992) Statement on Exercise. Benefits and recommendations for physical activity programs for all Americans. A statement for health professionals by the Committee on Exercise and Cardiac Rehabilitation of the Council on Clinical Cardiology, American Heart Association. *Circulation* 86:340-344.

Fogelman K, Fox AJ and Power C (1989) Class and tenure mobility: do they explain social inequalities in health among young adults in Britain? *In:* Fox J (ed) *Health inequalities in European countries*. Gower, Aldershot. p. 333-352.

Fox J, Goldblatt P and Jones D (1990) Social class mortality differentials: artifact, selection or life circumstances? *In:* Goldblatt P (ed) *Longitudinal Study 1971-1981: mortality and social organisation. Series LS No. 6*. HMSO, London. p. 100-108.

Frankel EN, Kanner J, German JB, Parks E and Kinsella JE (1993) Inhibition of oxidation of human low-density lipoprotein by phenolic substances in red wine. *Lancet* 341:454-457.

Freeman ZS (1990) Stress and hypertension - a critical review. *Med J Aust* 153:621-625.

Friedman GD and Klatsky AL (1993) Is alcohol good for your health? *N Engl J Med* 329:1882-1883.

Garrison RJ, Gold RS, Wilson PWF and Kannel WB (1993) Educational attainment and coronary heart disease risk: The Framingham Offspring Study. *Prev Med* 22:54-64.

Garrow JS (1992) Treatment of obesity. *Lancet* 340:409-413.

Gibberd RW, Dobson AJ, Florey C du V and Leeder SR (1984) Differences and comparative declines in ischaemic heart disease mortality among subpopulations in Australia, 1969-1978. *Int J Epidemiol* 13:25-31.

Gill JS, Zezulka AV, Shipley MJ, Gill SK and Beevers DG (1986) Stroke and alcohol consumption. *N Engl J Med* 315:1041-1046.

Glantz SA and Parmley WW (1991) Passive smoking and heart disease. Epidemiology, physiology, and biochemistry. *Circulation* 83:1-12.

Goldblatt P (1990) Mortality and alternative social classifications. *In:* Goldblatt P (ed) *Longitudinal Study 1971-1981: mortality and social organisation. Series LS. No. 6*. HMSO, London. p. 163-192.

Goldman L and Cook EF (1984) The decline in ischemic heart disease mortality rates. An analysis of the comparative effects of changes in medical interventions and changes in lifestyle. *Ann Intern Med* 101:825-836.

Gortmaker SL, Must A, Perrin JM, Sobel AM and Dietz WH (1993) Social and economic consequences of overweight in adolescence and young adulthood. *N Engl J Med* 329:1008-1012.

Gregory J, Foster K, Tyler H and Wiseman M (1990) *The dietary and nutritional survey of British adults. A survey of the dietary behaviour, nutritional status and blood pressure of adults aged 16 to 64 living in Great Britain*. Office of Population Censuses and Surveys, Social Survey Division, HMSO, London.

Gronbaek M, Deis A, Sorensen TIA, Becker U, Schnohr P, Jensen G (1995) Mortality associated with moderate intakes of wine, beer, or spirits. *Br Med J* 310:1165-1169.

Haan M, Kaplan GA and Camacho T (1987) Poverty and health. *Am J Epidemiol* 125:989-998.

Hamm P, Shekelle RB and Stamler J (1989) Large fluctuations in body weight during young adulthood and twenty-five-year risk of coronary death in men. *Am J Epidemiol* 129:312-318.

Hammar N, Alfredsson L and Theorell T (1994) Job characteristics and the incidence of myocardial infarction. *Int J Epidemiol* 23:277-284.

Hanna E, Dufour MC, Elliott S, Stinson F and Harford TC (1992) Dying to be equal: women, alcohol, and cardiovascular disease. *Br J Addict* 87:1593-1597.

Hardes GR, Dobson AJ, Lloyd DM and Leeder SR (1985) Coronary heart disease mortality trends and related factors in Australia. *Cardiology* 72:23-28.

Hart N (1986) Inequalities in health: The individual versus the environment. *J R Statist Soc A* 149:228-246.

Hasan J (1989) Way-of-life, stress and differences in morbidity between occupational classes. *In:* Fox J (ed) *Health inequalities in European countries*. Gower, Aldershot. p. 372-385.

Haskell WL, Leon AS, Caspersen CJ, et al. (1992) Cardiovascular benefits and assessment of physical activity and physical fitness in adults. *Med Sci Sports Exerc* 24(Suppl.):S201-S220.

Heller RF, Rose G, Tunstall-Pedoe HD and Christie DGS (1978) Blood pressure measurement in the United Kingdom Heart Disease Prevention Project. *J Epidemiol Community Health* 32:235-238.

Heller RF, Dobson AJ, Alexander HM, Steele PL and Malcolm JA (1992) Changes in drug treatment and case fatality of patients with acute myocardial infarction. Observations from the Newcastle MONICA Project, 1984/1985 to 1988/1990. *Med J Aust* 157:83-86.

Helmert U, Shea S, Herman B and Greiser E (1990) Relationship of social class characteristics and risk factors for coronary heart disease in West Germany. *Public Health* 104:399-416.

Helmrich SP, Ragland DR, Leung RW and Paffenbarger RS Jr (1991) Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. *N Engl J Med* 325:147-152.

Hendriks HF, Veenstra J, Velthuis-te Wierik EJM, Schaafsma G and Kluft C (1994) Effect of moderate dose of alcohol with evening meal on fibrinolytic factors. *Br Med J* 308:1003-1006.

Hennekens CH, Peto R, Hutchison GB and Doll R (1988) An overview of the British and American aspirin studies. *N Engl J Med* 318:923-924.

Henry AA, Cully LR, Grigson T and Lee C (1991) Recent trends in the prescribing of cholesterol lowering drugs in Australia. *Med J Aust* 155:332-336.

Hense HW, Kuulasmaa K, Zaborskis A, Kupsc W and Tuomilehto J (1990) Quality assessment of blood pressure measurements in epidemiological surveys. The impact of last digit preference and the proportions of identical duplicate measurements. *Rev Epidemiol Sante Publique* 38:463-468.

Hense HW (1991) Inaccuracy of the Hawksley random zero sphygmomanometer. *Lancet* 337:556.

Hessel PA (1986) Terminal digit preference in blood pressure measurements: effects on epidemiological associations. *Int J Epidemiol* 15:122-125.

Hetzel BS , Charnock JS , Dwyer T and McLennan PL (1989) Fall in coronary heart disease mortality in USA and Australia due to sudden death: evidence for the role of polyunsaturated fat. *J Clin Epidemiol* 42:885-893.

Higgins M, D'Agostino R, Kannel W and Cobb J (1993) Benefits and adverse effects of weight loss. Observations from the Framingham Study. *Ann Intern Med* 119:758-763.

Hill DJ, White VM, Pain MD and Gardner GJ (1990) Tobacco and alcohol use among Australian secondary schoolchildren in 1987. *Med J Aust* 152:124-130.

Hill DJ, White VM and Gray NJ (1991) Australian patterns of tobacco smoking in 1989. *Med J Aust* 154:797-801.

Hill DJ, White VM, Williams RM and Gardner GJ (1993) Tobacco and alcohol use among Australian secondary school students in 1990. *Med J Aust* 158:228-234.

Himes JH, Bouchard C and Pheley AM (1991) Lack of correspondence among measures identifying the obese. *Am J Prev Med* 7:107-111.

Hockey R, Hobbs MST and Martin CA (1991) Improvement in survival following myocardial infarction in Perth 1971-1988 [abstract]. *Aust NZ J Med* 21:553.

Hodge RL (1984) Risk factors in Australians: National Heart Foundation's risk factor prevalence study, 1980. *Aust NZ J Med* 14:395-399.

Holme I, Helgeland A, Hjermann I and Lund-Larsen PG (1976) Coronary risk factors and socioeconomic status. The Oslo study. *Lancet* ii:1396-1398.

Holme I, Helgeland A, Hjermann I, Leren P and Lund-Larsen PG (1980) Four-year mortality by some socioeconomic indicators: the Oslo study. *J Epidemiol Community Health* 34:48-52.

Holtzman NA (1989) General comments of conference participants. *In:* Bunker JP, Gomby DS, Kehrer BH (eds) *Pathways to health. The role of social factors.* The Henry J Kaiser Family Foundation, California. p. 235-241.

Hopkins A, Margetts BM, Cohen J and Armstrong BK (1980) Dietary change among Italians and Australians in Perth. *Community Health Stud* 4:67-75.

Hurley SF, Williams SL and McNeil JJ (1990) Trends in prescribing of antihypertensive drugs in Australia, 1977-1987. *Med J Aust* 152:259-266.

Hypertension Detection and Follow-up Program Cooperative Group (1978) Variability of blood pressure and the results of screening in the Hypertension Detection and Follow-up Program. *J Chron Dis* 31:651-667.

Illsley R (1955) Social class selection and class differences in relation to stillbirths and infant deaths. *Br Med J* 2:1520-1524.

Illsley R (1986) Occupational class, selection and the production of inequalities in health. *Q J Socl Aff* 2:151-165.

Illsley R and Le Grand J (1993) Regional inequalities in mortality. *J Epidemiol Community Health* 47:444-449.

Jackson R, Beaglehole R, Yee RL, Small C and Scragg R (1990) Trends in cardiovascular risk factors in Auckland, 1982 to 1987. *NZ Med J* 103:363-365.

Jackson R, Scragg R and Beaglehole R (1991) Alcohol consumption and risk of coronary heart disease. *Br Med J* 303:211-216.

Jackson R, Scragg R and Beaglehole R (1992) Does recent alcohol consumption reduce the risk of acute myocardial infarction and coronary death in regular drinkers? *Am J Epidemiol* 136:819-24.

Jacobs DR Jr, Mebane IL, Bangdiwala SI, Criqui MH and Tyroler HA (1990) High density lipoprotein cholesterol as a predictor of cardiovascular disease mortality in men and women: the follow-up study of the Lipid Research Clinics Prevalence Study. *Am J Epidemiol* 131:32-47.

Jacobsen BK and Thelle DS (1988) Risk factors for coronary heart disease and level of education. The Tromso Heart Study. *Am J Epidemiol* 127:923-932.

Jalkanen L, Tuomilehto J, Nissinen A and Puska P (1989) Changes in body mass index in a Finnish population between 1972 and 1982. *J Intern Med* 226:163-170.

Jamrozik K and Hockey R (1989) Trends in risk factors for vascular disease in Australia. *Med J Aust* 150:14-18.

Jarjoura D and Logue E (1990) Variation in heart disease mortality across census tracts as a function of overdispersion and social class mixture. *Stat Med* 9:1199-1209.

Jeffery RW (1991) Population perspectives on the prevention and treatment of obesity in minority populations. *Am J Clin Nutr* 53 (suppl):1621-1624.

Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (1993) The fifth report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (JNC V). *Arch Intern Med* 153:154-183.

Jones IG and Cameron D (1984) Social class analysis - an embarrassment to epidemiology. *Community Med* 6:37-46.

Jupp J (1990) Two hundred years of immigration. *In:* Reid J and Trompf P (eds) *The health of immigrant Australia. A social perspective*. Harcourt Brace Jovanovich, Sydney. p. 1-38.

Kannel WB (1988) Alcohol and cardiovascular disease. *Proc Nutr Soc* 47:99-110.

Kannel WB (1991) Epidemiology of essential hypertension: the Framingham experience. *Proc R Coll Physicians Edinb* 21:273-287.

Kannel WB, Cupples LA, Ramaswami R, Stokes J III, Kreger BE and Higgins M (1991) Regional obesity and risk of cardiovascular disease; The Framingham Study. *J Clin Epidemiol* 44:183-190.

Kaplan GA and Keil JE (1993) Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation* 88:1973-1998 (Special report).

Karasek R, Baker D, Marxer F, Ahlbom A and Theorell T (1981) Job decision latitude, job demands, and cardiovascular disease: a prospective study of Swedish men. *Am J Public Health* 71:694-705.

Kaufman DW, Rosenberg L, Helmrich SP and Shapiro S (1985) Alcoholic beverages and myocardial infarction in young men. *Am J Epidemiol* 121:548-554.

Keys A, Menotti A, Karvonen MJ, et al. (1986) The diet and 15-year death rate in the seven countries study. *Am J Epidemiol* 124:903-915.

Kirk-Gardner R and Crossman J (1991) Cardiac risk factors of smoking, hypertension, obesity and family history: a review of the literature. *Can J Cardiovasc Nurs* 2:9-14.

Kitagawa EM and Hauser PM (1973) *Differential mortality in the United States: A study in socioeconomic epidemiology*. Harvard University Press, Cambridge.

Klag MJ, Ford DE, Mead LA, et al. (1993) Serum cholesterol in young men and subsequent cardiovascular disease. *N Engl J Med* 328:313-318.

Klatsky AL, Armstrong MA and Friedman GD (1992) Alcohol and mortality. *Ann Intern Med* 117:646-654.

Kohl HW III, Powell KE, Gordon NF, Blair SN and Paffenbarger RS Jr (1992) Physical activity, physical fitness, and sudden cardiac death. *Epidemiol Rev* 14:37-58.

Koskinen S (1985) *Time trends in cause-specific mortality by occupational class in England and Wales*. Paper presented to the IUSSP Conference, Florence.

Kraus JF, Borhani NO and Franti CE (1980) Socioeconomic status, ethnicity, and risk of coronary heart disease. *Am J Epidemiol* 111:407-414.

Krupinski J (1984) Changing patterns of migration to Australia and their influence on the health of migrants. *Soc Sci Med* 18:927-937.

Kuczmarski RJ (1992) Prevalence of overweight and weight gain in the United States. *Am J Clin Nutr* 55(Suppl 2):495S-502S.

Kushnar RF (1993) Body weight and mortality. *Nutr Rev* 51:127-136.

Kuskowska-Wolk A and Bergström R (1993a) Trends in body mass index and prevalence of obesity in Swedish men 1980-89. *J Epidemiol Community Health* 47:103-108.

Kuskowska-Wolk A and Bergström R (1993b) Trends in body mass index and prevalence of obesity in Swedish women 1980-89. *J Epidemiol Community Health* 47:195-199.

Lakka TA, Venäläinen JM, Rauramaa R, Salonen R, Tuomilehto J and Salonen JT (1994) Relation of leisure-time physical activity and cardiorespiratory fitness to the risk of acute myocardial infarction in men. *N Engl J Med* 330:1549-1554.

LaRosa JC, Hunninghake D, Bush D, et al. (1990) The cholesterol facts. A summary of the evidence relating dietary fats, serum cholesterol, and coronary heart disease. A joint statement by the American Heart Association and the National Heart, Lung, and Blood Institute. *Circulation* 81:1721-1733.

Larsson B (1992) Obesity and body fat distribution as predictors of coronary heart disease. *In:* Marmot M and Elliott P (eds) *Coronary heart disease epidemiology. From aetiology to public health*. Oxford University Press, New York. p. 233-241.

Law MR, Wald NJ, Wu T, Hackshaw A and Bailey A (1994a) Systematic underestimation of association between serum cholesterol concentration and ischaemic heart disease in observational studies: data from the BUPA study. *Br Med J* 308:363-366.

Law MR, Wald NJ and Thompson SG (1994b) By how much and how quickly does reduction in serum cholesterol concentration lower risk of ischaemic heart disease? *Br Med J* 308:367-373.

Leclerc A (1989) Differential mortality by cause of death: a comparison between selected European countries. *In:* Fox J (ed) *Health inequalities in European countries.* Gower, Aldershot.

Lee C (1993) Definition and assessment of physical activity. *Am J Public Health* 17:190-194.

Lee I-M, Manson JE, Hennekens CH and Paffenbarger RS Jr (1993) Body weight and mortality. A 27-year follow-up of middle-aged men. *JAMA* 270:2823-2828.

Leeder SR, Dobson AJ, Gibberd RW and Lloyd DM (1984) Declining mortality rates from ischemic heart disease in Australia. *Aust NZ J Med* 14:388-394.

Leon AS, Connett J, Jacobs DR Jr, Rauramad R (1987) Leisure-time physical activity levels and risk of coronary heart disease and death. *JAMA* 258:2388-2395.

Lester IH (1994) *Australia's food and nutrition.* AGPS, Canberra. p. 58-70.

Liberatos P, Link BG and Kelsey JL (1988) The measurement of social class in epidemiology. *Epidemiol Rev* 10:87-121.

Lindenstrøm E, Boysen G and Nyboe J (1994) Influence of total cholesterol, high density lipoprotein cholesterol. and triglycerides on risk of cerebrovascular disease: the Copenhagen city heart study. *Br Med J* 309:11-15.

Lissner L, Odell PM, D'Agostino RB, et al. (1991) Variability of body weight and health outcomes in the Framingham population. *N Engl J Med* 324:1839-1844.

Loos CA and Halais CM (1991) Waist:hip ratio versus body mass index - screening for risk of cardiovascular disease. *Aust J Nutr Diet* 48:113-117.

Lovell RRH and Prineas RJ (1974) Differences in blood pressure measurements and prevalence of hypertension between Australian-born and Italian-born middle-aged men and women in Melbourne. *Med J Aust* 2:893-898.

Luepker RV, Rosamond WD, Murphy R, et al. (1993) Socioeconomic status and coronary heart disease risk factor trends. The Minnesota Heart Survey. *Circulation* 88:2172-2179.

Lynch JW, Kaplan GA, Cohen RD, et al. (1994) Childhood and adult socioeconomic status as predictors of mortality in Finland. *Lancet* 343:524-527.

Lynch P and Oelman BJ (1981) Mortality from CHD in the British army compared with the civil population. *Br Med J* 283:405-407.

Lynge E, Anderson O and Lars-Gunnar H (1989) Mortality: a comparison of within-country differentials based on selected occupational groups reported in a variety of countries. *In:* Fox J (ed) *Health inequalities in European countries.* Gower, Aldershot. p. 163-172.

Macintyre S (1986) The patterning of health by social position in contemporary Britain: directions for sociological research. *Soc Sci Med* 23:393-415.

Macintyre S (1989) The role of health services in relation to inequalities in health in Europe. *In:* Fox J (ed) *Health inequalities in European countries.* Gower, Aldershot. p. 317-332.

Macklin J (1990) *The National Health Strategy: setting the agenda for change. Background paper No. 1.* National Health Strategy Unit, Melbourne. p. 18.

MacMahon S, Peto R, Cutler J, et al. (1990) Blood pressure, stroke, and coronary heart disease. Part 1, prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. *Lancet* 335:765-774.

Manderson L and Reid JC (1994) What's culture got to do with it? *In:* Waddell C and Petersen AR (eds) *Just health. Inequality in illness, care and prevention.* Churchill Livingstone, Melbourne.

Manninen V, Tenkanen L, Koskinen P, Huttunen JK, Manttari M, Heinonen OP and Frick MH (1992) Joint effects of serum triglyceride and LDL cholesterol concentrations on coronary heart disease risk in the Helsinki Heart Study. *Circulation* 85:37-45.

Manson JE, Stampfer MJ, Hennekens CH and Willett WC (1987) Body weight and longevity: a reassessment. *JAMA* 257:353-358.

Manson JE, Rimm EB, Stampfer MJ, et al. (1991a) Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. *Lancet* 338:774-778.

Manson JE, Colditz GA, Stampfer MJ, et al. (1991b) A prospective study of maturity-onset diabetes mellitus and risk of coronary heart disease and stroke in women. *Arch Intern Med* 151:1141-1147.

Manson JE, Tosteson H, Satterfield S, et al. (1992a) The primary prevention of myocardial infarction. *N Engl J Med* 326:1406-1416.

Manson JE, Nathan DM, Krolewski AS, Stampfer MJ, Willett WC and Hennekens CH (1992b) A prospective study of exercise and incidence of diabetes among US male physicians. *JAMA* 268:63-67.

Margetts BM, Hopkins SM, Binns CW, Miller MR and Armstrong BK (1981) Nutrient intakes in Italian migrants and Australians in Perth. *Food Nutr* 38:7-10.

Marmot MG and Morris JN (1984) The Social Environment. *In:* Holland W, Detels R and Knox G (eds) *Oxford Textbook of Public Health Volume 1. History, Determinants, Scope and Strategies.* Oxford University Press, Oxford. p. 97-118.

Marmot MG, Shipley MJ and Rose G (1984) Inequalities in death-specific explanations of a general pattern? *Lancet* 1:1003-1006.

Marmot MG (1986) Social inequalities in mortality: the social environment. *In:* Wlkinson RG (ed) *Class and health research and longitudinal data.* Tavistock, London. p.21-33.

Marmot MG and McDowall ME (1986) Mortality decline and widening social inequalities. *Lancet* 2:274-276.

Marmot MG, Kogevinas M and Elston MA (1987) Social/economic status and disease. *Annu Rev Publ Health* 8:111-135.

Marmot MG and Theorell T (1988) Social class and cardiovascular disease: the contribution of work. *Int J Health Serv* 18:659-674.

Marmot MG (1989) Socioeconomic determinants of CHD mortality. *Int J Epidemiol* 18(Suppl 1):S196-S202.

Marmot M and Brunner E (1991) Alcohol and cardiovascular disease: the status of the U shaped curve. *Br Med J* 303:565-568.

Martin CA, Hobbs MST and Armstrong BK (1984) The fall in mortality from ischemic heart disease in Australia: has survival after myocardial infarction improved? *Aust NZ J Med* 14:435-438.

Martin CA, Hobbs MST and Armstrong BK (1987) Estimation of myocardial infarction mortality from routinely collected data in Western Australia. *J Chron Dis* 40:661-669.

Martin CA, Hobbs MST, Armstrong BK and De Klerk NH (1989) Trends in the incidence of myocardial infarction in Western Australia between 1971 and 1982. *Am J Epidemiol* 129:655-668.

Martin FIR, Hopper JL and Dean B (1984) Glucose tolerance and mortality in diabetes mellitus in Maltese–born residents of Victoria. *Med J Aust* 141:93-97.

Mathers C (1994a) *Health differentials among adult Australians aged 25–64 years. Australian Institute of Health and Welfare: Health Monitoring Series, No. 1.* AGPS, Canberra.

Mathers C (1994b) *Health differentials among older Australians.* Australian Institute of Health and Welfare: Health Monitoring Series No. 2, AGPS, Canberra.

McKeown T (1976) *The role of medicine: dream, mirage or nemesis.* Nuffield Provincial Hospitals Trust, London.

McMichael AJ and Hartshorne JM (1980) Cardiovascular disease and cancer mortality in Australia, by occupation, in relation to drinking, smoking and eating. *Community Health Stud* 4:76-84.

McMichael AJ and Hartshorne JM (1982) Mortality risks in Australian men by occupational groups, 1968-1978. *Med J Aust* 1:253-256.

McMichael AJ (1985) Social class (as estimated by occupational prestige) and mortality in Australian males in the 1970s. *Community Health Stud* 9:220-230.

McMichael AJ (1989) Coronary heart disease: interplay between changing concepts of aetiology, risk distribution and social strategies for prevention. *Community Health Stud* 13:5-13.

McQueen DV and Siegrist J (1982) Social factors in the etiology of chronic disease: an overview. *Soc Sci Med* 16:353-367.

Meade TW, Chakrabarti R, Haines AP, North WRS and Stirling Y (1979) Characteristics affecting fibrinolytic activity and plasma fibrinogen concentrations. *Br Med J* 1:153-156.

Menotti A, Keys A, Blackburn H, et al. (1990) Twenty-year stroke mortality and prediction in twelve cohorts of the Seven Countries Study. *Int J Epidemiol* 19:309-315.

Miller GJ, Beckles GLA, Maude GH and Carson DC (1990) Alcohol consumption: protection against coronary heart disease and risks to health. *Int J Epidemiol* 19:923-930.

Milne R and Vessey M (1992) The pill and mortality - an overview. *J Public Health Med* 14:9-16.

Mitchell PI, Morgan MJ, Boadle DJ, et al. (1980) Role of alcohol in the aetiology of hypertension. *Med J Aust* 2:198-200.

Mittleman MA, Maclure M, Tofler GH, Sherwood JB, Goldberg RJ and Muller JE (1993) Triggering of acute myocardial infarction by heavy physical exertion. *N Engl J Med* 329:1677-1683.

Morris JN (1990) Inequalities in health: Ten years and little further on. *Lancet* 336:491-493.

Mueller WH, Wear ML, Hanis CL, et al. (1991) Which measure of body fat distribution is best for epidemiologic research? *Am J Epidemiol* 133:858-869.

Mullen K (1992) A question of balance: health behaviour and work context among male Glaswegians. Soc Hlth Illn 14:72-97.

Muller JE and Tofler GH (1991) Circadian variation and cardiovascular disease. *N Engl J Med* 325:1038-1039.

Multiple Risk Factor Intervention Trial Research Group (1982) Multiple risk factor intervention trial: risk factor changes and mortality results. *JAMA* 248:1465-1477.

Najman JM (1978) *A social epidemiology of Australia, using mortality data: 1965-67. PhD thesis.* University of New South Wales, Sydney.

Najman JM (1988) The measurement of socioeconomic inequality and social class in Australia: a review of past practices and recent developments. *Community Health Stud* 12:31-41.

Najman JM (1993) Health and poverty: past, present and prospects for the future. *Soc Sci Med* 36:157-166.

Najman JM (1994) Class inequalities in health and lifestyle. *In:* Waddell C and Petersen AR (eds) *Just health. Inequality in illness, care and prevention.* Churchill Livingstone, Melbourne.

National Campaign Against Drug Abuse (1992) *Statistics on drug abuse in Australia 1992.* AGPS, Canberra.

National Center for Health Statistics, Drizd T, Dannenberg AL and Engel A (1986) Blood pressure levels in persons 18-74 years of age in 1976-80, and trends in blood pressure from 1960 to 1980 in the United States. *Vital and Health Statistics* 11:67-68.

National Health and Medical Research Council (1987a) *The role of exercise in nutrition and health. 104th session, November 1987.* National Health and Medical Research Council, Canberra.

National Health and Medical Research Council (1987b) *Is there a safe level of daily consumption of alcohol for men and women? Recommendations regarding responsible drinking behaviour.* AGPS, Canberra.

National Health and Medical Research Council (1992a) *Dietary guidelines for Australians.* AGPS, Canberra.

National Health and Medical Research Council (1992b) *The role of polyunsaturated fats in the Australian diet. Report of the NHMRC Working Party.* AGPS, Canberra. pp. 27-34.

National Health Strategy (1992a) *Enough to make you sick. How income and environment affect health. Research Paper No. 1.* National Health Strategy Unit, Melbourne.

National Health Strategy (1992b) *Supplement to Research Paper No 1. Enough to make you sick. How income and environment affect health.* National Health Strategy Unit, Melbourne.

National Heart Foundation of Australia (1982a) Appendix I. Adjustment of blood pressure readings in Melbourne. *In: Risk Factor Prevalence Study. No 1 - 1980.* National Heart Foundation of Australia, Canberra.

National Heart Foundation of Australia (1982b) *Risk factor prevalence study, No. 1 - 1980.* NHFA, Canberra.

National Heart Foundation of Australia (1985) *Risk factor prevalence study, No. 2 - 1983.* NHFA, Canberra.

National Heart Foundation of Australia (1991a) *Exercise and heart disease. Policy statement No. 5.* NHFA, Canberra.

National Heart Foundation of Australia (1991b) *Alcohol and cardiovascular disease. Policy Statement No. 1.* NHFA, Canberra.

National Heart Foundation of Australia (1991c) *Make it part of your day [leaflet]. Advice from the heart [leaflet]. So you want to get more exercise [pamphlet].* NHFA, Canberra.

National Heart Foundation of Australia (1994a) *Cardiac surgery. Report No. 30.* NHFA, Canberra.

National Heart Foundation of Australia (1994b) *Coronary angioplasty 1992. Report No. 8.* NHFA, Canberra.

National Heart Foundation of Australia (1994c) *Heart Facts Report 1992.* NHFA, Canberra.

Neaton JD, Blackburn H, Jacobs D, et al. (1992) Serum cholesterol level and mortality findings for men screened in the Multiple Risk Factor Intervention Trial . *Arch Intern Med* 152:1490-1500.

Neaton JD and Wentworth D (1992) Serum cholesterol, blood pressure, cigarette smoking, and death from coronary heart disease. Overall findings and differences by age for 316,099 white men. *Arch Intern Med* 152:56-64.

Negri E, La Vecchia C, D'Avanzo B, Nobili A and La Malfa RG (1994) Acute myocardial infarction: association with time since stopping smoking in Italy. *J Epidemiol Community Health* 48:129-133.

NIH Consensus Development Panel on Triglyceride, High-Density Lipoprotein, and Coronary Heart Disease (1993) Triglyceride, high-density lipoprotein, and coronary heart disease. *JAMA* 269:506-510.

NIH Technology Assessment Conference Panel (1993) Methods for voluntary weight loss and control. *Ann Intern Med* 119:764-770.

Oberman A, Kuller LH and Carleton RA (1994) Prevention of cardiovascular disease - opportunities for progress. *Prev Med* 23:727-732.

O'Brien E, Mee F, Atkins N and O'Malley K (1990) Inaccuracy of the Hawksley random zero sphygmomanometer. *Lancet* 336:1465-1468.

Oldenburg B, Bauman A, Booth M and Owen N (1991) Increasing levels of physical activity in the Australian community. *Health Promot J Aust* 1:15-18.

Owen N and Bauman A (1992) The descriptive epidemiology of a sedentary lifestyle in adult Australian. *Int J Epidemiol* 21:305-310.

Padfield PL, Jyothinagaram SG, Watson DM, Donald P and McGinley IM (1990) Problems in the measurement of blood pressure. *J Hum Hypertens* 4(Suppl 2):3-7.

Paffenbarger RS Jr, Wing AL, Hyde RT and Jung DL (1983) Physical activity and incidence of hypertension in college alumni. *Am J Epidemiol* 117:245-257.

Paffenbarger RS Jr, Jung DL, Leung RW and Hyde RT (1991) Physical activity and hypertension: an epidemiological view. *Ann Med* 23:319-327.

Pamuk E (1985) Social class inequality in mortality from 1921 to 1972 in England and Wales. *Popul Stud* 39:17-31.

Paneth N (1994) The impressionable fetus? Fetal life and adult health. *Am J Public Health* 84:1372-1374.

Paneth N and Susser M (1995) Early origin of coronary heart disease (the 'Barker hypothesis'). *Br Med J* 310:411-412.

Pappas G, Queen S, Hadden W and Fisher G (1993) The increasing disparity in mortality between socioeconomic groups in the United States, 1960 and 1986. *N Engl J Med* 329:103-109.

Parker D, Liu K, Dyer AR, Giumetti D, Liao Y and Stamler J (1988) A comparison of the random-zero and standard mercury sphygmomanometers. *Hypertension* 11:269-272.

Pearson TA and Pyorala K (1989) Trends in CHD in selected countries: an overview. *Int J Epidemiol* 18(Suppl 1):S99-S100.

Pekkanen J, Linn S, Heiss G, et al. (1990) Ten-year mortality from cardiovascular disease in relation to cholesterol level among men with and without pre-existing cardiovascular disease. *N Engl J Med* 322:1700-1707.

Pekkanen J, Nissinen A, Vartiainen E, Salonen JT, Punsar S and Karvonen M (1994) Changes in serum cholesterol level and mortality: a 30-year follow-up. The Finnish Cohorts of the Seven Countries Study. *Am J Epidemiol* 139:155-165.

Phillimore P, Beattie A and Townsend P (1994) Widening inequality of health in northern England, 1981-91. *Br Med J* 308:1125-1128.

Pieper C, La Croix AZ and Karasek RA (1989) The relation of psychosocial dimensions of work with coronary heart disease risk factors: a meta-analysis of five United States data bases. *Am J Epidemiol* 129:483-494.

Pi-Sunyer FX (1991) Health implications of obesity. *Am J Clin Nutr* 53:1595S-1603S.

Pi-Sunyer FX (1993a) Medical hazards of obesity. *Ann Intern Med* 119:655-660.

Pi-Sunyer FX (1993b) Short-term medical benefits and adverse effects of weight loss. *Ann Intern Med* 119:722-726.

Pocock SJ, Shaper AG, Cook DG, Phillips AN and Walker M (1987) Social class differences in ischaemic heart disease in British men. *Lancet* 197-201.

Pocock SJ, Shaper AG and Phillips AN (1989) Concentrations of high density lipoprotein cholesterol, triglycerides, and total cholesterol in ischaemic heart disease. *Br Med J* 298:998-1002.

Polednak AP (1989) *Racial and ethnic differences in disease*. Oxford University Press, Oxford.

Powell KE, Thompson PD, Caspersen CJ and Kendrick JS (1987) Physical activity and the incidence of coronary heart disease. *Annu Rev Publ Health* 8:253-287.

Power C, Manor O, Fox AJ and Fogelman K (1990) Health in childhood and social inequalities in health in young adults. *J R Statist Soc A* 153:17-28.

Power C, Manor O and Fox J (1991) *Health and class: the early years*. Chapman & Hall, London.

Powles J and Birrell R (1977) *Mortality in Victoria 1969-1973*. Department of Sociology, Monash University, Melbourne.

Powles J, Ktenas D, Sutherland C and Hage B (1988) Food habits in Southern-European migrants: a case-study of migrants from the Greek island of Levkada. *In:* Truswell AS and Wahlqvist ML (eds) *Food habits in Australia*. Heinemann, Melbourne. p. 201-223.

Powles J and Salzberg M (1989) Work, class or life-style? Explaining inequalities in health. *In:* Lupton GM and Najman JM (eds) *Sociology of health and illness. Australian readings*. MacMillan, South Melbourne.

Powles J and Gifford S (1990) How healthy are Australia's immigrants? *In:* Reid J and Trompf P (eds) *The health of immigrant Australia. A social perspective*. Harcourt Brace Jovanovich, Orlando, Florida. p. 77-107.

Powles J, Hage B and Cosgrove M (1990) Health-related expenditure patterns in selected migrant groups: data from the Australian Household Expenditure Survey, 1984. *Community Health Stud* 14:1-7.

Powles JW and Gifford S (1993) Health of nations: lessons from Victoria, Australia. *Br Med J* 306:125-127.

Prineas RJ, Folsom AR and Kaye SA (1993) Central adiposity and increased risk of coronary artery disease mortality in older women. *Ann Epidemiol* 3:35-41.

Quine S, Lancaster PAL (1989) Indicators of social class - relationship between prestige of occupation and suburb of residence. *Community Health Stud* 13:510-518.

Razay G, Heaton KW, Bolton CH and Hughes AO (1992) Alcohol consumption and its relation to cardiovascular risk factors in British women. *Br Med J* 304:80-83.

Reader R (1972) Incidence and prevalence of ischaemic heart disease in Australia. *Med J Aust* 2(Suppl):3-6.

Reader R (1979) Heart disease in Australia 1960-1980 and the National Heart Foundation. *Med J Aust* 1:323-328.

Reaven PD, Barrett-Connor E and Edelstein S (1991) Relation between leisure-time physical activity and blood pressure in older women. *Circulation* 83:559-565.

Regan TJ (1990) Alcohol and the cardiovascular system. *JAMA* 264:377-381.

Renaud S and De Lorgeril M (1992) Wine, alcohol, platelets, and the French paradox for coronary heart disease. *Lancet* 339:1523-1526.

Research Group ATS-RF2-OB43 of the Italian National Research Council (1987) Time trends of some cardiovascular risk factors in Italy. *Am J Epidemiol* 126:95-103.

Reynes J, Lasater TM, Feldman H, Assaf AR and Carleton RA (1993) Education and risk factors for coronary heart disease: results from a New England community. *Am J Prev Med* 9:365-371.

Rich-Edwards JW, Manson JE, Hennekens CH and Buring JE (1995) The primary prevention of coronary heart disease in women. *New Eng J Med* 332:1758-1766.

Rimm EB, Giovannucci EL, Willett WC, et al. (1991) Prospective study of alcohol consumption and risk of coronary disease in men. *Lancet* 338:464-468.

Risk Factor Prevalence Study Management Committee (1990) *Risk Factor Prevalence Study: Survey No 3 1989*. National Heart Foundation of Australia and Australian Institute of Health, Canberra.

Rissanen A, Knekt P, Heliövaara M, Aromaa A, Reunanen A and Maatela J (1991) Weight and mortality in Finnish women. *J Clin Epidemiol* 44:787-795.

Robbins AS, Manson JE, Lee I-M, Satterfield S and Hennekens CH (1994) Cigarette smoking and stroke in a cohort of U.S. male physicians. *Ann Intern Med* 120:458-462.

Robinson RV and Kelley J (1979) Class as conceived by Marx and Dahrendorf: effects on income, inequality and politics in the U.S. and G.B. *Am Sociol Rev* 44:38-58.

Roncari DAK (1992) Obesity and coronary heart disease. *Can Med Assoc J* 146:525.

Rose G (1965) Standardisation of observers in blood-pressure measurement. *Lancet* 1:673-674.

Rose G (1985) Cardiovascular diseases. *In:* Holland W, Detels R and Knox G (eds) *Oxford Textbook of Public Health Volume 4. Specific Applications*. Oxford University Press, Oxford. p. 133-144.

Rose GA, Holland WW and Crowley EA (1964) A sphygmomanometer for epidemiologists. *Lancet* 1:296-300.

Russell M, Cooper ML, Frone MR and Welte JW (1991) Alcohol drinking patterns and blood pressure. *Am J Public Health* 81:452-457.

Salonen JT, Puska P, Tuomilehto J and Homan K (1982) Relation of blood pressure, serum lipids, and smoking to the risk of cerebral stroke. A longitudinal study in Eastern Finland. *Stroke* 13:327-333.

Salonen JT, Slater JS, Tuomilehto J and Rauramaa R (1988) Leisure time and occupational physical activity: risk of death from ischemic heart disease. *Am J Epidemiol* 127:87-94.

Sandvik L, Erikssen J, Thaulow E, Erikssen G, Mundal R and Rodahl K (1993) Physical fitness as a predictor of mortality among healthy, middle-aged Norwegian men. *N Engl J Med* 328:533-537.

Sexton PT, Jamrozik K and Walsh JM (1992) Death certification and coding for ischaemic heart disease in Tasmania. *Aust NZ J Med* 22:114-118.

Shaper AG, Pocock SJ, Walker M, Phillips AN, Whitehead TP and Macfarlane PW (1985) Risk factors for ischaemic heart disease: the prospective phase of the British Regional Heart Study. *J Epidemiol Community Health* 39:197-209.

Shaper AG (1988) *Coronary heart disease risks and reasons*. Current Medical Literature Ltd, London. p. 22-40.

Shaper AG (1990) Alcohol and mortality: a review of prospective studies. *Br J Addict* 85:837-847.

Shaper AG, Phillips AN, Pocock SJ, Walker M and Macfarlane PW (1991) Risk factors for stroke in middle aged British men. *Br Med J* 302:1111-1115.

Shaper AG and Wannamethee G (1991) Physical activity and ischaemic heart disease in middle-aged British men. *Br Heart J* 66:384-394.

Shaper AG, Wannamethee G and Walker M (1994) Alcohol and coronary heart disease: a perspective from the British Regional Heart Study. *Int J Epidemiol* 23:482-494.

Shinton R and Beevers G (1989) Meta-analysis of relation between cigarette smoking and stroke. *Br Med J* 298:789-794.

Shinton R, Shipley M and Rose G (1991) Overweight and stroke in the Whitehall study. *J Epidemiol Community Health* 45:138-142.

Shinton R and Sagar G (1993) Lifelong exercise and stroke. *Br Med J* 307:231-234.

Shrapnel WS, Calvert GD, Nestel PJ and Truswell AS (1992) Diet and coronary heart disease. *Med J Aust* 156(Suppl):S9-S16.

Shryock RH (1979) *The development of modern medicine: an interpretation of the social and scientific factors involved*. Victor Gollancz, London. p. 176-204.

Siegel PZ, Brackbill RM and Heath GW (1995) The epidemiology of walking for exercise: implications for promoting activity among sedentary groups. *Am J Public Health* 85:706-709.

Siegrist J, Siegrist K and Weber I (1986) Sociological concepts in the etiology of chronic disease: the case of ischemic heart disease. *Soc Sci Med* 22:247-253.

Siegrist J (1991) Contributions of sociology to the prediction of heart disease and their implications for public health. *Eur J Public Health* 1:10-21.

Simons LA, Simons J, Magnus P and Bennett SA (1986) Education level and coronary risk factors in Australians. *Med J Aust* 145:446-450.

Siskind V, Copeman R and Najman JM (1987) Socioeconomic status and mortality: A Brisbane area analysis. *Community Health Stud* 11:15-23.

Siskind V, Najman JM and Veitch C (1992) Socioeconomic status and mortality revisited: an extension of the Brisbane area analysis. *Aust J Public Health* 16:315-320.

Sjöström LV (1992a) Mortality of severely obese subjects. *Am J Clin Nutr* 55(Suppl 2):S516-S523.

Sjöström LV (1992b) Morbidity of severely obese subjects. *Am J Clin Nutr* 55(Suppl 2):S508-S515.

Slattery ML and Jacobs DR Jr (1988) Physical fitness and cardiovascular disease mortality. The US Railroad Study. *Am J Epidemiol* 127:571-580.

Smith A and Jacobson B (eds) (1988) *The nation's health: a strategy for the 1990s*. Kings' Fund Publishing Office, London. p. 107.

Smith GD, Bartley M and Blane D (1990) The Black report on socioeconomic inequalities in health 10 years on. *Br Med J* 301:373-377.

Smith GD, Shipley MJ, Marmot MG and Rose G (1992) Plasma cholesterol concentration and mortality. The Whitehall study. *JAMA* 267:70-76.

Sniderman A, Michel C and Racine N (1992) Heart disease in patients with diabetes mellitus. *J Clin Epidemiol* 45:1357-1370.

Sobolski J, Kornitzer M, De Backer G, et al. (1987) Protection against ischemic heart disease in the Belgian Physical Fitness Study: physical fitness rather than physical activity? *Am J Epidemiol* 125:601-610.

Sprafka JM, Burke GL, Folsom AR, Luepker RV and Blackburn H (1990) Continued decline in cardiovascular disease risk factors: results of the Minnesota Heart Survey, 1980-1982 and 1985-1987. *Am J Epidemiol* 132:489-500.

Stampfer MJ, Colditz GA, Willett WC, Speizer FE and Hennekens CH (1988) A prospective study of moderate alcohol consumption and the risk of coronary heart disease and stroke in women. *N Engl J Med* 319:267-273.

Stampfer MJ and Colditz GA (1991) Estrogen replacement therapy and coronary heart disease: a quantitative assessment of the epidemiologic evidence. *Prev Med* 20:47-63.

Steenland K (1992) Passive smoking and the risk of heart disease. *JAMA* 267:94-99.

Stehbens WE (1990) The epidemiological relationship of hypercholesterolemia, hypertension, diabetes mellitus and obesity to coronary heart disease and atherogenesis. *J Clin Epidemiol* 43:733-741.

Stenhouse NS and McCall MG (1970) Differential mortality from cardiovascular disease in migrants from England and Wales, Scotland and Italy, and native-born Australians. *J Chron Dis* 23:423-431.

Stern J (1983) Social mobility and the interpretation of social class mortality differentials. *J Soc Policy* 12:27-49.

Stewart MJ and Padfield PL (1992) Blood pressure measurement: an epitaph for the mercury sphygmomanometer? *Clin Sci* 83:1-12.

Strong PM (1990) Black on class and mortality: theory, method and history. *J Public Health Med* 12:168-180.

Stunkard AJ and Sorensen TIA (1993) Obesity and socioeconomic status - a complex relation. *N Engl J Med* 329:1036-1037.

Suh I, Shaten BJ, Cutler JA and Kuller LH for the Multiple Risk Factor Intervention Group (1992) Alcohol use and mortality from coronary heart disease: the role of high-density lipoprotein cholesterol. *Ann Intern Med* 116:881-887.

Syme SL, Berkman LF (1976) Social class, susceptibility and sickness. *Am J Epidemiol* 104:1-8.

Taskforce on Cardiovascular Disease (1986) Cardiovascular disease: preventing an unnecessary way of death. *In:* Better Health Commission (ed) *Looking forward to better health. Volume 2.* AGPS, Canberra.

Taylor R, Herrman H and Preston G (1983) *Occupation and mortality in Australia. Working age males, 1975-77.* Health Commission of Victoria and the Department of Social and Preventive Medicine, Monash University, Melbourne.

Teasdale TW, Sorensen TIA and Stunkard AJ (1990) Genetic and early environmental components in sociodemographic influences on adult body fatness. *Br Med J* 300:1615-1618.

The Committee on Diet and Heart Disease of the National Heart Foundation of Australia (1974) Dietary fat and coronary heart disease: a review. 1. Diet in the prevention of coronary heart disease and some pathogenetic aspects. *Med J Aust* 1:575-579.

The Coronary Prevention Group (1992) *Prevention of Cardiovascular Disease. Recommendations for National Governments from Technical Reports of the World Health Organisation*. World Health Organization, London.

The Royal College of Physicians (1991) *Medical aspects of exercise. Benefits and risks*. The Royal College of Physicians of London, London.

The Toronto Working Group on Cholesterol Policy (1990) Asymptomatic hypercholesterolemia: a clinical policy review. Chapter 2. Serum cholesterol and other risk factors for coronary heart disease. *J Clin Epidemiol* 43:1035-1051.

Thom TJ (1989) International mortality from heart disease: rates and trends. *Int J Epidemiol* 18(Suppl 1):S20-S28.

Thompson PL, Parsons RW, Jamrozik K, Hockey RL, Hobbs MST and Broadhurst RJ (1992) Changing patterns of medical treatment in acute myocardial infarction. Observations from the Perth MONICA Project 1984-1990. *Med J Aust* 157:87-92.

Thomson PL, Hobbs MST and Martin CA (1988) The rise and fall of ischemic heart disease in Australia. *Aust NZ J Med* 18:327-337.

Townsend P (1990a) Widening inequalities of health in Britain: A rejoinder to Rudolph Klein. *Int J Health Serv* 20:363-372.

Townsend P (1990b) Individual or social responsibility for premature death? Current controversies in the British debate about health. *Int J Health Serv* 20:373-392.

Tunstall-Pedoe H (1991) The Dundee coronary risk-disk for management of change in risk factors. *Br Med J* 303:744-747.

Turrell G, Western JS and Najman JM (1994) The measurement of social class in health research: problems and prospects. *In:* Waddell C and Petersen AR (eds) *Just health. Inequality in illness, care and prevention.* Churchill Livingstone, Melbourne.

Ulman R and Abernethy JD (1975) Blood pressure and length of stay in Australia of Italian immigrants in the Australian national blood pressure study. *Int J Epidemiol* 4:213-215.

U.S. Department of Health and Human Services (1983) *The health consequences of smoking: cardiovascular disease. A report of the Surgeon General 1983*. Public Health Service, Centers for Disease Control, Office on Smoking and Health, Rockville, Maryland.

U.S. Department of Health and Human Services (1990) Smoking cessation and cardiovascular disease. *In: The health benefits of smoking cessation. A report of the Surgeon General 1990.* Public Health Service, Centers for Disease Control, Office on Smoking and Health. DHHS Publication No. (CDC)90-8416, Rockville, Maryland. p. 187-273.

Ueshima H, Ozawa H, Baba S, et al. (1992) Alcohol drinking and high blood pressure: data from a 1980 national cardiovascular survey of Japan. *J Clin Epidemiol* 45:667-673.

Valkonen T (1989a) Appendix 7A: The calculation of the values of inequality coefficients and their changes. *In:* Fox J (ed) *Health inequalities in European countries.* Gower, Aldershot. p. 161-162.

Valkonen T (1989b) Adult mortality and level of education: a comparison of six countries. *In:* Fox J (ed) *Health inequalities in European countries.* Gower, Aldershot. p. 142-162.

Wadsworth MEJ (1986) Serious illness in childhood and its association with later-life achievement. *In:* Wilkinson RG (ed) *Class and health: research and longitudinal data.* Tavistock, London. p. 50-74.

Wakabayashi K, Nakamura K, Kono S, Shinchi K and Imanishi K (1994) Alcohol consumption and blood pressure: an extended study of self-defence officials in Japan. *Int J Epidemiol* 23:307-311.

Wannamethee G and Shaper AG (1992) Physical activity and stroke in British middle aged men. *Br Med J* 304:597-601.

Wannamethee SG, Shaper AG, Whincup PH and Walker M (1995) Smoking cessation and the risk of stroke in middle-aged men. *JAMA* 274:155-160.

Webb K and Manderson L (1990) Food habits and their influence on health. *In:* Reid J and Trompf P (eds) *The health of immigrant Australia. A social perspective.* Harcourt Brace Jovanovich, Sydney. p. 154-205.

West P (1991) Rethinking the health selection explanation for health inequalities. *Soc Sci Med* 32:373-384.

Whelton PK (1994) Epidemiology of hypertension. *Lancet* 344:101-106.

Whitehead M (1987) *The health divide: inequalities in health in the 1980s.* Health Education Council, London.

Whitehead M, Judge K, Hunter DJ, Maxwell R and Scheuer MA (1993) Tackling inequalities in health: the Australian experience. *Br Med J* 306:783-787.

WHO MONICA Project, Keil U and Kuulasmaa K (1989) WHO MONICA Project: Risk factors. *Int J Epidemiol* 18(Suppl 1):S46-S55.

WHO MONICA Project Principal Investigators (prepared by H Tunstall-Pedoe) (1988) The World Health Organization MONICA Project (Monitoring trends and determinants in cardiovascular disease): A major international collaboration. *J Clin Epidemiol* 41:105-114.

Wilcox J (1960) Observer factors in the measurement of blood pressure. *Nursing Res* 10:4-17.

Wilhelmsen L (1990) Synergistic effects of risk factors. *Clin Exp Hypertens* A12:845-863.

Wilkins PS (1992) Medicinal virtues of alcohol in moderation. *Med J Aust* 156:807-808.

Wilkinson RG (1986) Socio-economic differences in mortality: interpreting the data on their size and trends. *In:* Wilkinson RG (ed) *Class and health: research and longitudinal data.* Tavistock, London. p. 1-20.

Wilkinson RG (1992) Income distribution and life expectancy. *Br Med J* 304:165-168.

Willett WC, Green A, Stampfer MJ, et al. (1987) Relative and absolute excess risks of coronary heart disease among women who smoke cigarettes. *N Engl J Med* 317:1303-1309.

Williamson DF and Pamuk ER (1993) The association between weight loss and increased longevity. A review of the evidence. *Ann Intern Med* 119:731-736.

Willich SN, Lewis M, Löwel H, Arntz H-R, Schubert F and Schröder R (1993) Physical exertion as a trigger of acute myocardial infarction. *N Engl J Med* 329:1684-1690.

Wilson AF (1993) Methods and applications of genetic analysis in the study of cardiovascular disease: a summary of contributions to GAW 8. *Genetic Epidemiol* 10:503-512.

Wing S, Hayes C, Heiss G, et al. (1986) Geographic variation in the onset of decline of ischemic heart disease mortality in the United States. *Am J Public Health* 76:1401-1408.

Wing S, Dargent-Molina P, Casper M, Riggan W, Hayes C and Tyroler HA (1987) Changing association between community occupational structure and ischaemic heart disease mortality in the United States. *Lancet* ii:1067-1070.

Wing S, Casper M, Riggan W, Hayes C and Tyroler HA (1988) Socioenvironmental characteristics associated with the onset of decline of ischemic heart disease mortality in the United States. *Am J Public Health* 78:923-926.

Winkleby MA, Fortmann SP and Barrett DC (1990) Social class disparities in risk factors for disease: eight-year prevalence patterns by level of education. *Prev Med* 19:1-12.

Winkleby MA, Jatulis DE, Frank E and Fortmann SP (1992) Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. *Am J Public Health* 82:816.

Winkleby MA (1994) The future of community-based cardiovascular disease intervention studies. *Am J Public Health* 84:1369-1372

Witteman JCM, Willett WC, Stampfer MJ, et al. (1990) Relation of moderate alcohol consumption and risk of systemic hypertension in women. *Am J Cardiol* 65:633-637.

Wood PD, Stefanick ML, Dreon DM, et al. (1988) Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise. *N Engl J Med* 319:1173-1179.

Woodward M, Shewry MC, Smith WCS and Tunstall-Pedoe H (1992) Social status and coronary heart disease: results from the Scottish Heart Health Study. *Prev Med* 21:136-148.

World Health Organization (1986) *MONICA manual, Version 1.1, December 1986, CVD/MNC*. World Health Organization, Geneva.

World Health Organization (1992) *MONICA manual, Part III: Population survey, March 1992*. World Health Organization, Geneva.

Yao C-H, Slattery ML, Jacobs DR Jr, Folsom AR and Nelson ET (1991) Anthropometric predictors of coronary heart disease and total mortality: findings from the US Railroad Study. *Am J Epidemiol* 134:1278-1289.

Yeracaris CA and Kim JH (1978) Socioeconomic differentials in selected causes of death. *Am J Public Health* 68:342-351.

Young CM (1986a) Differential mortality of birthplace groups in Australia during 1980-82. *J Aust Popul Assoc* 3:144-168.

Young CM (1986b) *Selection and survival. Immigrant mortality in Australia.* AGPS and Department of Immigration and Ethnic Affairs, Canberra.

Young CM (1987) Migration and mortality: The experience of birthplace groups in Australia. *Int Migration Rev* 21:531-554.

Young CM (1991) Changes in the demographic behaviour of migrants in Australia and the transition between generations. *Popul Stud* 45:67-89.

Young CM (1992a) Women's health, use of medical services, medication, lifestyle and chronic illness. Some findings from the 1989-90 National Health Survey. *In:* Donovan J, d'Espaignet E, Merton C and van Ommeren M (eds) *Immigrants in Australia: a health profile. Ethnic Health Series No. 1.* Australian Institute of Health and Welfare, AGPS, Canberra. p. 122-191.

Young CM (1992b) Mortality: the ultimate indicator of survival: the differential experience between birthplace groups. *In:* Donovan J, d'Espaignet E, Merton C and van Ommeren M (eds) *Immigrants in Australia: a health profile. Ethnic Health Series no. 1.* Australian Institute of Health and Welfare, AGPS, Canberra. p. 34-70.

Young CM (1992c) Australia's heterogeneous immigrant population: a review of demographic and socioeconomic characteristics. *In:* Donovan J, d'Espaignet E, Merton C and van Ommeren M (eds) *Immigrants in Australia: a health profile. Ethnic Health Series No. 1.* Australian Institute of Health and Welfare, AGPS, Canberra. p. 11-33.

Yusuf S, Wittes J and Friedman L (1988) Overview of results of randomised clinical trials in heart disease. II. Unstable angina, heart failure, primary prevention with aspirin, and risk factor modification. *JAMA* 260:2259-2263.

Zhang YQ, MacLennan R and Berry G (1984) Mortality of Chinese in New South Wales, 1969-1978. *Int J Epidemiol* 13:188-191.

Zimmet PZ, Collins VR, Dowse GK, et al. (1991) The relation of physical activity to cardiovascular disease risk factors in Mauritians. *Am J Epidemiol* 134:862-875.

Appendix A

Questionnaire (1989 survey)

IN CONFIDENCE

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NATIONAL HEART FOUNDATION RISK FACTOR STUDY 1989

1 5

6 11

Telephone number(s) where you may be contacted. Home _ _ _ _ _ _ _ _ _ _ _ _

Work _ _ _ _ _ _ _ _ _ _ _ _

12 17

CONSENT AND FORWARDING OF RESULTS

I consent to undergo the tests performed at the clinic and I understand that the results of my assessment will be given to me and/or my doctor if I wish.

18

I further understand that information and blood specimens collected in the course of the study will be used for research purposes, the results of which will be published in scientific journals or reports in such a way that individual participants cannot be identified.

I also understand that my own answers in this questionnaire and the results of my tests will not be released to anyone, even to my own doctor, without my specific permission.

8 9

Signature _ Date _ _ _ / _ _ _ / 1989.

19 24

To whom would you like your results sent?
(Please tick the appropriate box)

To no-one .... ☐

25

To myself only .... ☐

To my doctor only* .... ☐

26 27

To myself and my doctor* ☐

28 29

*If you want your results sent to your doctor, please write the name and address below.

Dr _

Address _

_ Postcode _ _ _ _ _ _

30

31 32

33 34

OFFICE USE ONLY

To be copied from page 12.

Weight _____kg

Height _____cm

Blood pressure:

Average systolic _____ mmHg

Average diastolic _____ mmHg

Total cholesterol _____mmol/l

H.D.L. cholesterol _____mmol/l

Triglycerides _____ mmol/l

Ferritin _____µg/l

Iron _____ µmol/l

Transferrin _____ µmol/l

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DIRECTIONS

- Please indicate your answer by ticking the appropriate box ☑ or by writing your answer in the space provided.
- Please use BLOCK LETTERS.
- If you are uncertain about the answer to any of the questions leave them blank and ask the receptionist to help you when you have reached the end of the questionnaire.
- Please do not write in the far right hand column of each page **(Office use only).**

☐☐☐☐☐ 1 5

☐☐☐☐☐☐ 6 11

1. Date of birth: ___ / ___ / **19**___
day mth year

☐☐☐☐☐☐ 12 17

2. Sex: Male............ ☐ 1
Female........ ☐ 2

☐ 18

3. Marital status:

Never married........................ ☐ 1
Now married.......................... ☐ 2
Separated but not divorced ☐ 3
Divorced................................ ☐ 4
Widowed .... ☐ 5

☐☐☐☐ 8 9 — 19 24

☐ 25

4. How many children and full-time students are living with you in your care?

None .... ☐
Children 0–14 years ____ number
Full-time students 15–24 years ____ number

26 ☐☐ 27
28 ☐☐ 29

5. Living arrangements:

Living with legal husband or wife .... ☐ 1
Living with partner as a couple (such as de facto marriage)..................... ☐ 2
Living with other person(s) (such as children, parents, flatmates) ☐ 3
Living alone .... ☐ 4

☐ 30

6. Where were you born? ________________________________
(Write State or Territory if born in Australia. Write country if born overseas.)

☐☐ 31 32

7. If you were not born in Australia, how many years have you lived in Australia? __________ years

☐☐ 33 34

Office use only

8. Please indicate the highest level of education you have completed.

Never attended school	☐ 1	
Primary school	☐ 2	
Some high school	☐ 3	
Completed high school (Year 12 or equivalent)	☐ 4	☐ 35
University, C.A.E. or other tertiary institution	☐ 5	

9. When did you last have your blood pressure measured?

In the last three months	☐ 1	
In the last six months	☐ 2	
In the last year	☐ 3	
In the last three years	☐ 4	
More than three years ago	☐ 5	
Never measured	☐ 6	☐ 36
Don't know	☐ 7	

10. When did you last have your blood cholesterol measured?

In the last three months	☐ 1	
In the last six months	☐ 2	
In the last year	☐ 3	
In the last three years	☐ 4	
More than three years ago	☐ 5	
Never measured	☐ 6	☐ 37
Don't know	☐ 7	

11. Have you ever been told that you have any of the following?

	No	Yes	
High blood pressure	☐ 1	☐ 2	☐ 38
Angina	☐ 1	☐ 2	☐ 39
Heart attack *(a "coronary", coronary occlusion, coronary thrombosis, myocardial infarction)*	☐ 1	☐ 2	☐ 40
Stroke	☐ 1	☐ 2	☐ 41
High cholesterol	☐ 1	☐ 2	☐ 42
High triglycerides	☐ 1	☐ 2	☐ 43

	No	Yes	
12. Are you on tablets for blood pressure?	☐ 1	☐ 2	☐ 44
13. Are you having treatment to lower your blood fat?	☐ 1	☐ 2	☐ 45
14. Are you on tablets or other treatment for angina?	☐ 1	☐ 2	☐ 46

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15. Has a doctor or nurse ever told you that you had diabetes?

No ☐ 1 Yes ☐ 2 — ☐ 47

If yes, please state the year you were first told 19 _ _ _
Year

☐☐ 48 49

16. Has a doctor or nurse ever told you that you showed sugar in the urine?

No ☐ 1 Yes ☐ 2 — ☐ 50

If yes, please state the year you were first told 19 _ _ _
Year

☐☐ 51 52

17. Have you ever been given advice or treatment for diabetes or sugar trouble?

No ☐ 1 Yes ☐ 2 — ☐ 53

If yes, please state the year this advice or treatment was first given 19 _ _ _
Year

☐☐ 54 55

Was this Diet advice ..... ☐ 1
Tablets ..... ☐ 2
Insulin injections ..... ☐ 3
Diet advice and tablets ..... ☐ 4
Diet advice and injections ..... ☐ 5

☐ 56

QUESTIONS 18 TO 21 FOR WOMEN ONLY

18. Have you ever taken the oral contraceptive pill?

Yes ..... ☐ 1
No ..... ☐ 2 **Go to Question 21.**

☐ 57

19. For how long altogether have you taken the oral contraceptive pill?
(Please estimate the total of all periods of use.)

Less than 6 months ..... ☐ 1
Between 6 months and 2 years ..... ☐ 2
Between 2 and 5 years ..... ☐ 3
Between 5 and 10 years ..... ☐ 4
Longer than 10 years ..... ☐ 5

☐ 58

20. Are you now taking the oral contraceptive pill?

Yes ..... ☐ 1
No ..... ☐ 2

☐ 59

21. Are you now pregnant?

Yes ..... ☐ 1
No ..... ☐ 2

☐ 60

In Questions 22 to 25 we want to find out about the exercise you had during the PAST 2 WEEKS;
- **For recreation, sport or health-fitness purposes,**
- **As part of your tasks at work and around the house.**

Please distinguish between vigorous exercise which made you breathe harder or puff and pant, and less vigorous exercise.

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RECREATION, SPORT OR HEALTH-FITNESS

22. In the PAST 2 WEEKS, did you engage in vigorous exercise — exercise which made you breathe harder or puff and pant? *(e.g. vigorous sports such as football, netball, tennis, squash, athletics; jogging or running; keep-fit exercises; vigorous swimming; etc.)*

No ..... ☐ 1
Yes..... ☐ 2

61

If yes, how many sessions of vigorous exercise did you have over the 2 week period? _ _ _ _ _ _ _ _ _

62 ☐☐ 63

Please estimate the TOTAL TIME spent exercising vigorously during the PAST 2 WEEKS. _ _ _ / _ _ _ _
hours minutes

64 ☐☐☐☐ 67

23. In the PAST 2 WEEKS, did you engage in less vigorous exercise for recreation, sport or health-fitness purposes which did not make you breathe harder or puff and pant?

No ..... ☐ 1
Yes..... ☐ 2

68

If yes, how many sessions of less vigorous exercise did you have over the 2 week period? _ _ _ _ _ _ _

69 70

24. In the PAST 2 WEEKS, did you walk for recreation or exercise?

No ..... ☐ 1
Yes..... ☐ 2

71

if yes, how many times? _ _ _ _ _ _ _

72 73

VIGOROUS TASKS AT WORK AND AROUND THE HOUSE (Paid or unpaid work)

25. In the PAST 2 WEEKS, did you engage in vigorous activity, apart from exercise, which made you breathe harder or puff and pant? *(e.g. carrying loads, heavy gardening, chopping wood, labouring — at home, during employment or anywhere else.)*

No ..... ☐ 1
Yes..... ☐ 2

74

If yes, how many sessions of these types of vigorous activity did you have over the 2 week period? _ _ _

75 76

Please estimate the TOTAL TIME spent in these types of vigorous activity during the PAST 2 WEEKS.

_ _ _ / _ _ _ _
hours minutes

77 ☐☐☐☐ 80

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26. Have you ever smoked cigarettes, cigars or a pipe regularly?

Yes.......... ☐ 1

No........... ☐ 2 **Go to Question 33.**

81

27. At what age did you start smoking regularly?

I started smoking regularly at _ _ _ _ years of age.

82 83

28. Have you given up smoking?

Yes, I gave up smoking in _ _ _ _ / 19 _ _ _
mth year

No, I still smoke............ ☐ 8888 **Go to Question 30.**

84 87

If you have GIVEN UP SMOKING please answer the following questions:

29. How much did you smoke?

I used to smoke _ _ _ _ _ manufactured cigarettes a **day** — 88 89

_ _ _ _ _ grams* "hand-rolled" cigarette tobacco per **week** — 90 92

_ _ _ _ _ cigars per **week** — 93 94

_ _ _ _ _ grams pipe tobacco per **week** — 95 97

If you CURRENTLY SMOKE please answer Questions 30 to 32; otherwise go to Question 33.

30. I currently smoke _ _ _ _ _ manufactured cigarettes a **day** — 98 99

_ _ _ _ _ grams* "hand-rolled" cigarette tobacco per **week** — 100 102

_ _ _ _ _ cigars per **week** — 103 104

_ _ _ _ _ grams pipe tobacco per **week** — 105 107

*NOTE: A 1¾ ounce pouch of cigarette tobacco equals 50 grams.

31. Which brand of manufactured cigarette do you usually smoke?
(Copy the name from a packet if possible)

I don't smoke manufactured cigarettes .... ☐ 995 **Go to Question 33.**

108 110

The brand I usually smoke is _
(Because of the number of varieties of many brands, please give this in full e.g. Benson and Hedges Extra Mild, Marlboro Red.)

32. Have you switched to lower tar manufactured cigarettes?

Yes, in _ _ _ _ / 19 _ _ _
mth year

No...................... ☐ 8888

I don't know........ ☐ 9999

111 114

33. How often do you usually drink alcohol?

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I don't drink alcohol	1	**Go to Question 35.**
Less than once a week	2	
On 1 or 2 days a week	3	
On 3 or 4 days a week	4	
On 5 or 6 days a week	5	
Every day	6	

115

34. On a day when you drink alcohol, how many drinks do you usually have?

1 or 2 drinks	1
3 or 4 drinks	2
5 to 8 drinks	3
9 to 12 drinks	4
13 to 20 drinks	5
More than 20 drinks	6

116

35. Do you add salt to your food after it is cooked?

Rarely or never	1
Sometimes	2
Almost always or always	3

117

36. Which of the following best describes your usual way of eating?
(Please tick one box only.)

No special diet	1	
Vegetarian	2	
Weight reduction diet	3	
Diabetic diet	4	
Fat modified diet to lower blood fat	5	
Other	6	Please specify _____

118

37. How often do you eat the fat on meat?

Usually	1
Sometimes	2
Rarely or never	3

119

38. How much of the following dairy products do you usually have IN A WEEK?

	Number in a week		
Full cream milk	_____ (litres)	120	121
Skim milk	_____ (litres)	122	123
Low fat milk	_____ (litres)	124	125
Yoghurt — plain or flavoured	_____ (small cartons)	126	127
Low fat yoghurt — plain or flavoured	_____ (small cartons)	128	129
Cream	_____ (tablespoons)	130	131
Ice-cream	_____ (scoops)	132	133

Office use only

39. How tall are you without shoes?

Centimetres _ _ _ _ _ _ _

or Feet / inches _ _ _ _/_ _ _

Don't know.............................. ☐ 999

134 136

40. How much do you weigh without clothes and shoes?

Kilograms _ _ _ _ _ _ _ _

or Stone / pounds _ _ _ /_ _ _

Don't know.............................. ☐ 999

137 139

Questions 41 to 46 ask about employment and income.

The answers to these questions play an important part in understanding the patterns of health in the Australian community.

For example, risk factors for heart disease are known to vary between different groups in the community.

Knowledge about these differences helps in providing the best health care.

41. Do you have a full-time or part-time job of any kind?
(Either for payment or profit, or unpaid work in a family business)

Yes......... ☐ 1

No.......... ☐ 2 **Go to Question 44.**

140

42. In your main job, what is your occupation?

- Give full title.

  For example, Civil Engineering Draftsman, Accounts Clerk, Fast Foods Cook, 1st Class Welder, Extruding Machine Operator, Coal Miner.

- Armed Services personnel state rank as well as occupation.

- Public Servants state official designation (e.g. ASO3) as well as occupation.

  Occupation _

43. What are the main tasks or duties that you usually perform in that occupation?

- Describe as fully as possible.

  For example, preparing drawings for dam construction, recording and paying accounts, cooking hamburgers and chips, welding of high pressure steam pipes, operating plastic extruding machine, operating continuous mining machine.

  Tasks or duties _

141 144

145 148

44. Which of the following describes the current employment status of yourself and of your partner (if applicable)?

- Here, partner means the person you are living with as legal husband or wife or 'de facto'.
- Please tick more than one box where applicable.

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	Self	Partner (spouse or 'de facto')	Office use only
Working full-time	☐ 1	☐ 1	☐ 149 ☐ 150
Working part-time	☐ 1	☐ 1	☐ 151 ☐ 152
Not working (but not retired)	☐ 1	☐ 1	☐ 153 ☐ 154
Home duties	☐ 1	☐ 1	☐ 155 ☐ 156
Full-time student	☐ 1	☐ 1	☐ 157 ☐ 158
Part-time student	☐ 1	☐ 1	☐ 159 ☐ 160
Retired	☐ 1	☐ 1	☐ 161 ☐ 162
Permanently unable to work/ill	☐ 1	☐ 1	☐ 163 ☐ 164
Other (please specify) _____			
_____	☐ 1	☐ 1	☐ 165 ☐ 166

45. What is the gross income of yourself and of your partner (if applicable)?

- Include income from all sources (e.g. wages, interest, pensions, Family Allowance Supplement and other benefits, tax rebates) before tax or anything else is taken out.
- Please estimate as best you can.

Gross income (i.e. before tax):	Self	Partner (spouse or 'de facto')
No income	☐ 1	☐ 1
$1 to $135 per week ($1 to $7,000 per year)	☐ 2	☐ 2
$136 to $173 per week ($7,001 to $9,000 per year)	☐ 3	☐ 3
$174 to $212 per week ($9,001 to $11,000 per year)	☐ 4	☐ 4
$213 to $250 per week ($11,001 to $13,000 per year)	☐ 5	☐ 5
$251 to $289 per week ($13,001 to $15,000 per year)	☐ 6	☐ 6
$290 to $327 per week ($15,001 to $17,000 per year)	☐ 7	☐ 7
$328 to $365 per week ($17,001 to $19,000 per year)	☐ 8	☐ 8
$366 to $404 per week ($19,001 to $21,000 per year)	☐ 9	☐ 9
$405 to $442 per week ($21,001 to $23,000 per year)	☐ 10	☐ 10
$443 to $577 per week ($23,001 to $30,000 per year)	☐ 11	☐ 11
$578 to $769 per week ($30,001 to $40,000 per year)	☐ 12	☐ 12
$770 to $962 per week ($40,001 to $50,000 per year)	☐ 13	☐ 13
Over $962 per week (Over $50,000 per year)	☐ 14	☐ 14

Office use only: ☐☐ 167 168 ☐☐ 169 170

46. What is the main source of income of yourself and of your partner (if applicable)?

	Self		Partner (spouse or 'de facto')	
Wages or salary	☐	1	☐	1
Own business or share in partnership	☐	2	☐	2
Any government pension or cash benefit	☐	3	☐	3
Superannuation	☐	4	☐	4
Investment / interest	☐	5	☐	5
Other (please specify) _	☐	6	☐	6

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☐ ☐
171 172

PLEASE STOP HERE

Thank you for your co-operation. Please now return this questionnaire to the receptionist and tell her about any difficulties you had with these questions.

OFFICE USE ONLY

To be completed during examination

Weight _____ kg 173 ☐☐☐ . ☐ 176

Height _____ cm 177 ☐☐☐ 179

Waist circumference:

1st reading _____ cm 180 ☐☐☐ 182

2nd reading _____ cm 183 ☐☐☐ 185

Hip (buttocks) circumference:

1st reading _____ cm 186 ☐☐☐ 188

2nd reading _____ cm 189 ☐☐☐ 191

Blood pressure observer _____ ☐ 192

Sphygmomanometer _____ 193 ☐☐ 194

Ambient temperature _____ °C 195 ☐☐ 196

Blood pressure:

1st reading:

Systolic _____ mm Hg 197 ☐☐☐ 199

Diastolic _____ mm Hg 200 ☐☐☐ 202

2nd reading:

Systolic _____ mm Hg 203 ☐☐☐ 205

Diastolic _____ mm Hg 206 ☐☐☐ 208

Have you had anything to eat or drink in the past 12 hours apart from water, black tea or black coffee?

No ☐ 1

Yes ☐ 2 ☐ 209

Have you donated blood in the last 12 months?

No ☐ 1

Yes ☐ 2 ☐ 210

If yes, how recently did you donate blood?

Less than 1 week ago..... ☐ 1

Between 1 week and 1 month ago..... ☐ 2

Between 1 month and 3 months ago ..... ☐ 3

Between 3 months and 6 months ago..... ☐ 4 ☐ 211

Between 6 months and 12 months ago..... ☐ 5

Do you regularly take iron supplements or a multi-vitamin mineral supplement containing iron?

No..... ☐ 1

Yes..... ☐ 2 ☐ 212

Don't know.... ☐ 3

Appendix B

Comparison of data items collected in each survey

TABLE A2.1 *Data items collected in the National Heart Foundation's Risk Factor Prevalence Study*

1980 survey			1983 survey			1989 survey		
Cities	**n**	**%**	**Cities**	**n**	**%**	**Cities**	**n**	**%**
Sydney North	702	69.6	Sydney North	1034	76.5	Sydney North	985	77.8
Sydney South	699	66.8	Sydney South	874	69.4	Sydney South	698	60.7
Melbourne	681	65.7	Melbourne	806	59.9	Melbourne	848	69.7
Brisbane	858	81.6	Brisbane	1004	76.2	Brisbane	815	67.1
Adelaide	901	82.7	Adelaide	1054	77.0	Adelaide	1935	72.8
Perth	901	87.4	Perth	1788	84.5	Perth	963	74.7
Hobart	875	77.1	Hobart	1080	77.7	Hobart	1084	83.4
						Darwin	1000	88.7
						Canberra	981	78.5
Total	5671	75.9	Total	7640	75.3	Total	9309	74.7
Sociodemographics			**Sociodemographics**			**Sociodemographics**		
Date of birth			Date of birth			Date of birth		
Sex			Sex			Sex		
Marital status			Marital status			Marital status		
						Children and full-time students		
						Living arrangements		
Place of birth			Place of birth			Place of birth		
- years in Australia			- years in Australia			- years in Australia		
Highest level of education			Highest level of education			Highest level of education		
Current employment status			Current employment status			Current employment status		
Occupation (CCLO)			Occupation (CCLO)			Occupation (ASCO)		
- hours worked each week			- hours worked each week					
						Gross income (self and partner)		
						Main source (self and partner)		
Conditions, treatment			**Conditions, treatment**			**Conditions, treatment**		
Pain or discomfort in chest?								
Pressure or heaviness in chest?								
Rose questionnaire								
Ever been told that you have:			Ever been told that you have:			Ever been told that you have:		
- high blood pressure			- high blood pressure			High blood pressure		
- angina pectoris			- angina pectoris			Angina		
- heart attack			- heart attack			Heart attack		
- stroke			- stroke			Stroke		
- high cholesterol			- high cholesterol			High cholesterol		
- high triglycerides			- high triglycerides			High triglycerides		
Treatment for blood pressure			Tablets for blood pressure			Tablets for blood pressure		
Treatment- lowering fat in blood			Treatment to lower blood fat			Treatment to lower blood fat		
			Tablets/other treatment-angina			Tablets/other treatment-angina		
Ever told you have diabetes?			Told had diabetes by Dr/nurse?			Told had diabetes by Dr/nurse?		
			-year first told			-year first told		
			Told sugar in urine?			Told sugar in urine?		
			-year first told			-year first told		
On tablets/injections for diabetes?			Given advice/treatment for diabetes or sugar trouble?			Given advice/treatment for diabetes or sugar trouble?		
			-year first told			-year first given		
			-advice/treatment			-advice/treatment		
Pill use (women only)			**Pill use (women only)**			**Pill use (women only)**		
Ever taken o/contraceptive pill			Ever taken o/contraceptive pill			Ever taken o/contraceptive pill		
Total duration of use			Total duration of use			Total duration of use		

Now taking o/contraceptive pill	Now taking o/contraceptive pill	Now taking o/contraceptive pill
Are you now pregnant	Are you now pregnant	Are you now pregnant
Sedatives, pain relievers etc		
How often do you take:		
Tranquillisers, sedatives		
Pain relievers eg aspirin, bex		
pills or medicine		
Sleep		
Hours usually sleep of a night		
Kind of sleep (poor, fair, good)		
How often do you take sleeping		
Dietary behaviour	**Dietary behaviour**	**Dietary behaviour**
How often do you take vitamins, tonics or mineral supplements	How often do you take vitamins, tonics or mineral supplements	
Add salt to food after cooking	Add salt to food after cooking	Add salt to food after cooking
	Usual way of eating (diet)	Usual way of eating (diet)
Fat modified diet for blood fat		
-year of commencement		
Diet to control weight		
-year of commencement		
Any other special diet		
-year of commencement		
-type of diet		
How often eat meat		
How often eat the fat on meat		How often eat the fat on meat
Eggs per week		
Spread used most		
How much dairy products:		How much dairy products:
Full cream milk		Full cream milk
Skim milk		Skim milk
Low fat milk		Low fat milk
Yogurt-plain or flavoured		Yogurt-plain or flavoured
Low fat yogurt		Low fat yogurt
Cream		Cream
Ice-cream		Ice-cream
Cheese		
Sugar with each hot drink-teaspoons		
Sugar with each bowl of cereal		
Alcohol use	**Alcohol use**	**Alcohol use**
Have you ever drunk alcohol		
How often usually drink alcohol	How often usually drink alcohol	How often usually drink alcohol
How many drinks usually have	How many drinks usually have	How many drinks usually have
How much low alcohol beer	How much low alcohol beer	
Smoking behaviour	**Smoking behaviour**	**Smoking behaviour**
Ever smoked regularly	Ever smoked regularly	Ever smoked regularly
-age started	-age started	-age started
-have you given up	-have you given up	-have you given up
-gave up smoking in month/year	-gave up smoking in month/year	-gave up smoking in month/year
IF GIVEN UP	IF GIVEN UP	IF GIVEN UP
Used to smoke:	Used to smoke:	Used to smoke:
Manuf cigs on a working day	Manufactured cigs a day	Manufactured cigs a day
Manuf cigs on a leisure day		
Hand-rolled per week (gms)	Hand-rolled per week (gms)	Hand-rolled per week (gms)
Small cigars per week	Cigars per week	Cigars per week
Large cigars per week		
Pipe tobacco per week (gms)	Pipe tobacco per week (gms)	Pipe tobacco per week (gms)
IF CURRENTLY SMOKE	IF CURRENTLY SMOKE	IF CURRENTLY SMOKE
Currently smoke:	Currently smoke:	Currently smoke:
Manuf cigs on a working day	Manufactured cigs a day	Manufactured cigs a day
Manuf cigs on a leisure day		

Hand-rolled per week (gms)	Hand-rolled per week (gms)	Hand-rolled per week (gms)
Small cigars per week	Cigars per week	Cigars per week
Large cigars per week		
Pipe tobacco per week (gms)	Pipe tobacco per week (gms)	Pipe tobacco per week (gms)
Usual brand	Usual brand	Usual brand
Switched to lower tar	Switched to lower tar	Switched to lower tar
-switched in month/year	-switched in month/year	-switched in month/year
Physical activity	**Physical activity**	**Physical activity**
	RECREATION, SPORT, HEALTH FITNESS	
How often do you engage in active exercise:		
	In past 2 weeks, did you:	In past 2 weeks, did you:
	Engage in vigorous exercise	Engage in vigorous exercise
	-no of sessions	-no of sessions
	-total time	-total time
Other sporting/ keep-fit exercise	Less vigorous exercise	Less vigorous exercise
	-no of sessions	-no of sessions
Walk for relaxation or exercise	Walk for recreation or exercise	Walk for recreation or exercise
	-no of times	-no of times
Any other physical exercise		
EMPLOYED FULL OR PART-TIME	TASKS AT WORK AND AROUND THE HOUSE (PAID OR UNPAID)	VIGOROUS TASKS AT WORK AND AROUND THE HOUSE (PAID OR UNPAID)
Time walking about whilst working	Involved in moderate to heavy physical exertion?	In past 2 weeks did you engage in vigorous activity apart from exercise?
Distance walked to and from work	- total time (at work)	- no of sessions
Freq. of cycling to and from work	- total time (around the house)	- total time
- distance travelled		
How much physical exertion at work		
Personality type (A/B)	**Personality type (A/B)**	
General Health Questionnaire	**General Health Questionnaire**	
Physical examination	**Physical examination**	**Physical examination**
Height	Height	Height
Weight	Weight	Weight
	Mid upper arm circumference	
		Waist circumference
		Hip circumference
	Sphygmomanometer	Sphygmomanometer
		Ambient temperature
	Blood pressure observer	Blood pressure observer
Systolic blood pressure	Systolic blood pressure	Systolic blood pressure
Diastolic blood pressure	Diastolic blood pressure	Diastolic blood pressure
Pulse rate		
Blood analysis	**Blood analysis**	**Blood analysis**
Fasting status	Fasting status	Fasting status
Cholesterol	Cholesterol	Cholesterol
HDL cholesterol	HDL cholesterol	HDL cholesterol
Triglyceride	Triglyceride	Triglyceride
	Glucose	
		Donated blood in last 12 months?
		- how recently
		- regularly take iron/ multi-vitamin supplement
		Ferritin
		Iron
		Transferin
		Transferin saturation